# Calcasieu Estuary Remedial Investigation/Feasibility Study (RI/FS): Baseline Ecological Risk Assessment (BERA)

## *Appendix H3: Assessment of Risks to Piscivorus Birds in the Calcasieu Estuary*

*Prepared For:*

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*Under Contract To:*

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*Prepared – October 2002 – By:*

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**CONTRACT NO. 68-W5-0022**
**DOCUMENT CONTROL NO. 3282-941-RTZ-RISKZ-14858**

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# Appendix H3. Assessment of Risks to Piscivorus Birds in the Calcasieu Estuary

## 1.0 Introduction

Development and industrialization in and around the Calcasieu Estuary in southwestern Louisiana in recent decades has led to concerns of environmental contamination in the area. A Remedial Investigation/Feasibility Study (RI/FS) was commissioned to determine the risks posed by environmental contamination to ecological receptors inhabiting key areas of the Calcasieu Estuary. A Baseline Ecological Risk Assessment (BERA) is required to meet this objective. This Appendix is part of the BERA and is conducted in accordance with the procedures laid out by the USEPA in the *Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessment* (USEPA 1997a). Under the eight-step process described by the USEPA for conducting a BERA, a screening ecological risk assessment (SERA) must first be conducted to determine preliminary estimates of exposure and risk.

The SERA for the Calcasieu Estuary (CDM 1999) identified areas of concern (AOCs), contaminants of concern (COCs), and ecological receptors potentially at risk. The SERA findings were revisited in a Baseline Problem Formulation (BPF) to yield a refined list of contaminants of concern, areas of interest, and ecological receptors to be considered in the BERA. The Phase II data collection provided more information and, therefore, a better tool to estimate risk at a screening level. Using this information, a conservative, deterministic assessment was conducted and can be

found in Appendix G along with a description of the methods used to identify COCs and areas of concern for piscivorus birds.

This Appendix is organized as follows. Section 1 provides a brief overview of the results of the conservative, deterministic ERA for wildlife described in detail in Appendix G. The AOCs and COCs that screened through the conservative, deterministic assessment for piscivorus birds are described in this section. Section 1 also includes a description of the conceptual model for piscivorus birds in the Calcasieu Estuary. A statement outlining the purpose of this assessment concludes Section 1.

Section 2 describes the probabilistic risk assessment methods used to estimate risks of COCs to piscivorus birds in the Calcasieu AOCs. Section 3 describes the probabilistic risk assessment results and Section 4 identifies the sources of uncertainty that could influence the estimated risks for piscivorus birds. The final section of this Appendix, Section 5, contains the conclusions regarding risks of COCs to piscivorus birds in the Calcasieu Estuary.

## 1.1 Deterministic Ecological Risk Assessment Summary

The methods and results of the deterministic ecological risk assessment are presented in detail in Appendix G. In summary, the deterministic assessment used a conservative approach to estimate risk to piscivorus birds from chemicals of potential concern (COPCs) in the Bayou d'Inde, Upper Calcasieu River, and Middle Calcasieu River Areas of Concern (BI AOC, UCR AOC, MCR AOC, respectively) of the Calcasieu Estuary system. Several reference sites, including Bayou Connine Bois and Choupique Bayou, were also included in the deterministic assessment to provide a

basis for comparison of risks. The deterministic assessment compared potentially attainable high exposures with conservative adverse effects benchmarks to provide a means of identifying which chemicals are a potential concern to piscivorus birds and in which areas of the Calcasieu Estuary system. A risk quotient (total daily intake/effect dose) for piscivorus birds greater than one, and greater than 1.2 times the risk quotient for the reference areas, for any COPC in any of the Calcasieu areas, resulted in the COPC being screened through to the probabilistic ecological risk assessment. COPCs that screened through the SERA are now referred to as contaminants of concern (COCs). Mercury was screened in for all three areas. TCDD and equivalents (TCDD-TEQs) screened in for the BI AOC and MCR AOC. Selenium and total PCBs only screened through in the BI AOC. The reference areas were included in the probabilistic risk assessment so that risks in the AOCs could be compared to background risks. Results of the deterministic risk assessment are presented in Table H3-1.

## 1.2 Contaminants of Concern

The COCs that screened through to the probabilistic risk assessment for piscivorus birds included TCDD-TEQs, selenium, mercury, and total PCBs. These COCs are described below.

***TCDD-TEQs***

Tetrachlorinated dibenzo-*p*-dioxins (TCDDs) and equivalents represent a group of aromatic compounds with similar properties (WHO 1989). The term equivalents refers to a specific group of polychlorinated dibenzo-*p*-dioxin (PCDDs) congeners, polychlorinated dibenzofuran (PCDFs) congeners and co-planar polychlorinated biphenyl (PCB) congeners. This group has a common structural relationship that

includes lateral halogenation and the ability to assume a planar conformation. The planar conformation is important as it leads to a common mechanism of action in many animal species that involves binding to the aryl hydrocarbon (Ah) receptor and elicitation of an Ah receptor-mediated biochemical and toxic response (van den Berg *et al.* 1998; Newsted *et al.* 1995; Safe 1994).

Each of these compounds, while similar in structure and acting at the same receptor, has different potencies, depending on the individual congener. To address these issues and effectively estimate the relative toxicity of these mixtures, a system has been created involving the development and use of toxic equivalency factors (TEFs). This approach is based on the *in vivo* and *in vitro* toxicity of each of the compounds in relation to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). 2,3,7,8-TCDD is considered to be the most toxic member of the this class of substances (van den Berg *et al.* 1998; Birnbaum and DeVito 1995; Safe 1994) and the toxicity of the others depends on the degree of chlorination, the chlorination sites, and the ability to achieve a planar form, relative to 2,3,7,8-TCDD. There are a number of assumptions made when using the TEF approach. These include: (1) the congeners are Ah-receptor antagonists and their toxicological potency is mediated by their binding affinity; and (2) no interaction occurs between the congeners and thus the sum of the individual congener effects accounts for the potency of the mixture. The overall effect of these assumptions is a potency estimate or toxic equivalent (TEQ) value. A more detailed discussion of the TEF approach for expressing the toxicity of this class of substances is presented in Appendix G.

The environmental degradation and metabolism of the congeners varies due to their unique physical/chemical properties. These can cause substantial differences between the congeners detected in environmental samples and the congener makeup of the original product (van den Berg *et al.* 1998). The majority of these congeners have

low solubility, low vapor pressure and high resistance to chemical breakdown, and are, therefore, highly persistent in the environment. They are also highly lipophilic with a high propensity to bind to organic and particulate matter. When released to aquatic systems, the majority of these compounds form associations with dissolved and/or particulate matter in the water column; biodegradation is considered to be a relatively minor fate process in water (NRCC 1981; Howard *et al.* 1991). Aquatic sediments provide a sink for these compounds and may represent long term sources to the aquatic food web (Kuehl *et al.* 1987; Muir *et al.* 1988; Corbet *et al.* 1983; Tsushimoto *et al.* 1982). As sediments are resuspended and carried downstream, they tend to accumulate in areas where currents are slow and the particles have time to settle.

Predatory organisms may be exposed to TCDD-TEQs through trophic transfer. PCDDs, PCDFs and PCB congeners are highly bioaccumulative substances that increase in concentration as they are passed up the food chain (i.e., biomagnification). For organisms inhabiting the Lake St. Clair ecosystem, Haffner *et al.* (1994) observed that PCB concentrations increased from 0.935 mg/kg in sediments, to 1.36 mg/kg in bivalves, to 7.24 mg/kg in oligochaetes, and to 64.9 mg/kg in predatory gar pike. Mink are particularly sensitive to PCBs and similar substances (Moore *et al.* 1999). Research has found that they accumulate PCBs in their subcutaneous fat at levels 38 to 200 times dietary concentrations, depending on the PCB congener (USEPA 1993). The avian predators of the Calcasieu estuary study area would similarly be expected to accumulate PCBs from the prey they consume.

This assessment estimates the risks posed by coplanar congeners to piscivorus birds because these compounds are expected to biomagnify up the food chain. Further, previous assessments have shown that species higher in the aquatic food chain are at particular risk of experiencing adverse effects, including reduced reproduction,

impaired growth and development, and death (Tillitt *et al.* 1992; Heaton *et al.* 1995). Piscivorus birds are high in the food chain and are potentially at high risk of exposure to coplanar congeners because they consume fish found in the Calcasieu Estuary system.

***Selenium***

The fate of selenium and its compounds in the environment is influenced to a large degree by its oxidation state. Changes in the valence state of selenium from -2 (hydrogen selenide) through 0 (elemental selenium), +2 (selenium dioxide), +4 (selenite) and +6 (selenate) affect the behavior of its different chemical compounds. The behavior of various compounds of selenium in the environment is also dependent on ambient conditions including pH, the presence of metal oxides and biological activity (ATSDR 1996; Maier *et al.* 1988).

Elemental selenium is essentially insoluble and will remain inert when released in the environment under anaerobic conditions. Heavy metal selenides and selenium sulfides predominate in acidic soils and soils with high organic matter, and will remain insoluble and immobile in this form (NAS 1976). Selenites and selenates are water soluble and are, therefore, more bioavailable in surface water and water contained in soils (Eisler 2000; ATDSR 1996; Robberecht and Van Grieken 1982). In general, these mobile forms of selenium dominate under aerobic and alkaline conditions. Sodium selenate is one of the most mobile selenium compounds in the environment because of its high water solubility and inability to adsorb onto particulates (NAS 1976). Selenium bioconcentrates and biomagnifies in aquatic food chains from invertebrates to birds (Ohlendorf *et al.* 1986a,b; Lemly 1985; Saiki and Lowe 1987; Saiki *et al.* 1993). Lemly (1985) reported BCFs of 1,500-1,850 and BAFs of 1,746-3,975 for selenium in freshwater species. These studies demonstrate

that selenium has the potential to biomagnify up the food chain and accumulate in piscivorus birds.

This assessment focuses on the risks posed by selenium to piscivorus birds because these compounds are expected to biomagnify up the food chain. Selenium bioconcentrates in aquatic food chains and diet is the primary source of exposure for piscivorus birds (Eisler 2000). Piscivorus birds are fairly high in the food chain and are potentially at high risk of exposure to selenium because they consume fish found in the Calcasieu Estuary.

***Mercury***

Mercury is found in the environment as the metal, $Hg^0$, and as divalent mercuric Hg(II) species. In the water column, $Hg^0$ is oxidized to Hg(II) under acidic conditions. Hg(II) undergoes a number of important reactions, one of which is methlylation by microbes and adsorption and absorption by biota (Stein *et al.* 1996). Biomethylation occurs both in the sediments, where sulfate-reducing bacteria are the primary methylators of mercury, and in the water column (Winfrey and Rudd 1990). Methylation in the water column also occurs abiotically, mediated by dissolved organic carbon (Weber 1993). Methylmercury may make up as much as 25 percent of the mercury in rivers and lakes (Gilmour and Henry 1991).

Methylmercury is highly soluble in water, extremely mobile, and thus readily enters the aquatic food web. Because methylation is higher under anaerobic conditions, benthic organisms in the anaerobic zones of sediment may be exposed to high methylmercury concentrations. These organisms are consumed by a variety of species, including piscivorus birds, leading to biomagnification up the food chain. The accumulation of methylmercury in aquatic organisms has been well documented, with concentrations in carnivorous fish 10,000 to >1,000,000 times the concentrations

found in ambient waters (Stein *et al.* 1996). Gilmour and Henry (1991) showed that fish from contaminated systems may continue to contain high levels of methylmercury long after inputs to the systems have ceased. Also, the efficient assimilation of the lipophilic methylmercury in fat and muscle and the lack of elimination results in increasing methylmercury concentrations with the age and size of fish and wildlife predators.

This assessment focuses on the risks posed by methylmercury to piscivorus birds because this species of mercury is more readily bioaccumulated and more toxic to wildlife than is metallic mercury. Further, previous assessments of methylmercury risks to wildlife have shown that species higher in the aquatic food chain are at particular risk of experiencing adverse effects, including reduced reproduction, impaired growth and development, and death (MacIntosh *et al.* 1994; USEPA 1997b; Moore *et al.* 1999). Piscivorus birds are high in the food chain and are potentially at high risk of exposure to mercury, because they consume fish found in BI AOC, UCR AOC, and MCR AOC.

***Total Polychlorinated Biphenyls (PCBs)***

Polychlorinated biphenyls (PCBs) is the generic term applied to mixtures of 209 chlorinated organic compounds that have similar molecular structures and properties. PCB congeners generally have low solubilities, low vapor pressure and high resistance to chemical breakdown. PCB mixtures are generally referred to in terms of the average degree of biphenyl chlorination (e.g., Aroclor 1254). Due to chemical stability, PCBs are highly persistent in the environment.

PCBs are persistent and highly lipophilic substances with low water solubility and a high propensity to bind to organic and particulate matter. In bulk releases to aquatic compartments, these substances will tend to remain as a non aqueous phase liquid and

settle to the bottom of the water body. Here, PCBs will gradually adsorb to organic and particulate matter and remain sequestered in sediment layers. Exposure to PCBs from this compartment occurs as a result of benthic organisms ingesting sediments during foraging and when sediments are stirred and PCB-laden particles are resuspended in the water column. As sediments are resuspended and carried downstream, they tend to accumulate in areas where currents are slow and the particles have time to settle.

Predatory organisms are exposed to PCBs primarily through trophic transfer. Organisms lower in the food chain may ingest and accumulate the substances, which are then passed on when they are consumed by higher food chain predators. Benthic communities are at the highest risk of direct exposure to PCBs. Benthic invertebrates will be exposed to PCBs through direct contact with interstitial pore water, ingestion of sediment particles, and ingestion of organisms that have also been exposed to substances. Pelagic organisms in the Calcasieu estuary will be exposed to PCBs through dermal and gill contact with surface waters; ingestion of water, suspended sediment, and organic matter; ingestion of sediment for bottom-feeding fish; and ingestion of other benthic and pelagic organisms. Uptake of PCBs by fish occurs mainly through the gills and the gastrointestinal tract (Shaw and Connell 1984). Most PCB accumulation in top fish predators can be attributed to the food pathway (Thomann 1989). Other species, such as amphibians, are also exposed to PCB-contaminated surface waters. Insectivorous, carnivorous, and piscivorus birds and mammals that reside, or partially reside, within the estuary are exposed to PCBs principally through diet and trophic transfer. PCBs are highly bioaccumulative substances that increase in concentration as they are passed up the food chain. For organisms inhabiting the Lake St. Clair ecosystem, Haffner *et al.* (1994) noted that PCB concentrations increased from 0.935 mg/kg in sediments, to 1.36 mg/kg in bivalves, to 7.24 mg/kg in oligochaetes, and to 64.9 mg/kg in predatory gar pike.

PCBs have also been shown to bioaccumulate in several bird species (Senthilkumar *et al.* 2001; Borga *et al.* 2001). The avian and mammalian predators of the Calcasieu Estuary study area would similarly be expected to accumulate PCBs from the prey they consume.

This assessment focuses on the risks posed by total PCBs to piscivorus birds because PCBs are expected to biomagnify up the food chain. Further, previous assessments of PCBs risks to wildlife have shown that species higher in the aquatic food chain are at particular risk of experiencing adverse effects, including reduced reproduction, impaired growth and development, and death (Moore *et al.* 1999). Piscivorus birds are fairly high in the food chain and are potentially at high risk of exposure to PCBs because they consume fish found in BI AOC.

## 1.3 Receptors of Concern

The exposure assessment for piscivorus birds exposed to TCDD-TEQs, selenium, mercury, and total PCBs, will be based on a hypothetical receptor that incorporates many of the characteristics typical of this receptor group. The characteristics of this hypothetical receptor are based upon the characteristics of six piscivorus birds potentially found in the Calcasieu Estuary: brown pelicans, belted kingfishers, osprey, least terns, Forster's terns, and Caspian terns. The following sections review the life histories and foraging behaviors of these six species. This information will then be used to create the life history and foraging behavior of a hypothetical receptor to be used in the assessment.

**Brown pelican *(Pelecanus occidentalis)***

The brown pelican is listed as endangered by the state of Louisiana and the United States. Brown pelicans are large stocky birds between 114 and 137 cm in height. They inhabit areas along the Gulf Coast, the Atlantic coast from North Carolina to Venezuela, and the Pacific coast from British Columbia to Chile (Knopf 1977). The average weight of adult males is usually greater than that of adult females, 3.7 kg compared to 3.2 kg (Hartman 1955). Adults have brown bodies and white heads. The most distinctive feature of the brown pelican is its large bill and throat pouch.

Brown pelicans are year round inhabitants of the Calcasieu Estuary. Brown pelicans nest in coastal areas. They prefer to nest on islands, which provide protection from predators. Nests are usually built on the ground, in mangrove trees, or in bushes (Knopf 1977). Brown pelicans are piscivorus birds, primarily fishing in shallow estuarine waters and rarely traveling further than 30 km out to sea. Adult brown pelicans require about 2 kg of fish per day to fulfill their dietary requirements. Young brown pelicans are fed by their parents for 9 weeks, consuming 70 kg of fish over that time (Arnqvist 1992).

Brown pelicans have a powerful stroking flight alternating with short glides. They fly with their head drawn back to the shoulder. Pelicans rarely soar and often fly just centimeters above the water. Small flocks of pelicans fly in long lines. Brown pelicans use their eyesight to spot schools of fish and then dive for them, sometimes completely submerging. The large pouch they have can hold up to 10 liters of water along with prey. The species of fish that brown pelicans most commonly eat are referred to as "rough" fish; meaning they are commercially unimportant. Brown pelicans consume the following "rough" fish species: menhaden, herring, sheepshead, pigfish, mullet, grass minnows, and silversides (Arnqvist 1992).

**Belted kingfisher *(Ceryle alcyon)***

Belted kingfishers are medium-sized birds averaging 33 cm in length. These birds are slate blue with a white neckband and belly. Females have a rust-colored band across their belly. Kingfishers have deep, irregular wingbeats, a relatively large head, and a loud, rattling call. They are commonly found throughout most of North America (USEPA 1993). Males and females are similar in size and body weight. A number of studies have reported mean body weights, including 136, 148, 150, and 158 g (USEPA 1993; 1995; Brooks and Davis 1987). Belted kingfishers are winter inhabitants in the Calcasieu region (Peterson 1980).

Preferred habitat is along stream, lake and pond edges, as well as sea coasts and estuaries (USEPA 1993). Because belted kingfishers are sight feeders, they prefer water that is clear and not obstructed by over hanging tree canopies or aquatic vegetation. Belted kingfishers typically nest in burrows in earthen banks beside rivers, streams, ponds and lakes. Belted kingfishers nest near suitable fishing areas when possible, but will nest away from water and feed in bodies of water other than the one closest to home. During spring and early summer, both male and female kingfishers defend a territory that includes both their nest site and their foraging area (USEPA 1993). By autumn, each bird defends an individual feeding territory only. Breeding territories are, on average, more than twice as long as non-breeding territories (e.g., 1,030 $\pm$ 219 m vs. 389.29 $\pm$ 92.63 m, respectively; USEPA 1993).

The belted kingfisher's diet primarily consists of fish and occasionally invertebrates (USEPA 1993). During shortages of their preferred foods, they will consume crabs, lizards, frogs, turtles, mussels, small snakes, insects, salamanders, newts, young birds, mice, and berries (USEPA 1993). Belted kingfishers typically forage on fish in the first 12 to 15 cm of the water column. This includes fish in shallow water, as well as fish in deeper water that swim close to the surface (USEPA 1993). Feeding

strategies vary depending upon the availability of perches that overhang the water. When adequate perches are available belted kingfishers will use them to spot prey in the water and then dive for them. Another technique is to fly over the surface of the water, waiting for potential prey to come into view, and then striking (USEPA 1993).

Belted kingfishers eat large fish relative to their body size (USEPA 1995). Several field studies have reported the size preferences of fish caught by belted kingfishers. The average length of fish caught in a field study in Michigan was less than 7.6 cm (range: 2.5 to 17.8 cm; Salyer and Lagler 1946). Another study found that belted kingfishers in Ohio selected fish ranging from 4 to 14 cm and 88% of the fish were between 6 and 12 cm (Davis 1982). The trophic level of the prey consumed by belted kingfishers varies slightly between regions. In a survey of field studies examining trophic level of belted kingfisher prey, USEPA (1995) found that 94% of prey were from the aquatic environment with trophic levels ranging between 2.6 and 3.

**Osprey *(Pandion haliaetus)***

Osprey are large birds of prey found world wide. The mean body weight of a female osprey is 1.57 kg, while the male is slightly smaller at 1.40 kg (USEPA 1993). Poole (1984) recorded body weights over different seasons; weights ranged from 1.42 to 1.93 kg in males and females. Body length ranged from 53 to 61 cm (USEPA 1993). It has a conspicuous crook in its long wings and a black wrist mark. The plumage is dark above, light below. In the past, osprey populations experienced sharp declines, as a result of DDT bioaccumulation and its impact on nesting success. More recently, the reduction or elimination of DDT in the environment, along with conservation efforts, have contributed to increasing the osprey population (NGS 1983). Osprey typically use southern regions, such as Louisiana, to overwinter (Peterson 1980; McLaren/Hart-Chemrisk 1998).

Osprey are predominantly found in marine environments, but will also live in large inland lakes, rivers, and estuaries if the habitat is suitable. Ideal nesting sites are found close to open, shallow water with an abundance of prey (USEPA 1993). They nest in large trees with an open crown, power poles and other man-made structures, and on artificial platforms. Osprey pairs are usually solitary, breed for life, and display strong nest site fidelity (USEPA 1993; USGS 2001b).

Osprey are primarily piscivorus birds. However, nearly all osprey will take other prey, such as birds, frogs, and crustaceans (USEPA 1993). Prey preferences change seasonally with abundance of local fish. If necessary, osprey will forage 10-15 km away from their nest (USEPA 1993). The osprey's position in the food chain makes it a good indicator of toxic substances that bioaccumulate (USEPA 1993). Osprey feed twice a day, in the mid-morning hours and again in late afternoon. Each meal is approximately 300 g (Poole 1989). They are most successful at catching medium sized, slow moving benthic feeding fish in shallow waters (e.g., fish length between 11-30 cm; USEPA 1993; USGS 2001b). All parts of the fish are consumed with bones and indigestible parts being eliminated in fecal pellets (USEPA 1993). These birds hover, often 15 to 46 m high, then suddenly plunge, sometimes going completely under the water.

**Least tern *(Sterna antillarum)***

Least terns are the smallest North American tern. They are common on the east, west, and Gulf Coasts, but less common in inland areas (McLaren Hart 1996; NOAA 2001). Interior populations (more than 80 km from the coast) are listed as endangered by the state of Louisiana and the United States. Least terns use the southern coast of Louisiana for breeding and summer grounds (Peterson 1980). They average 22 cm in length and have a wingspan of 51 cm. Body weight ranges from 39.0 to 47.6 g with a mean of 43.1 g (standard deviation; SD=2.12 g; Dunning 1984). Distinctive

characteristics of these birds include their very rapid wingbeats, short legs, and short, forked tail (Gough 1998). Least terns have a black cap, a white forehead, a yellow bill, and yellowish legs.

Least terns are a colonial ground nesting species. They prefer nesting on nearly bare ground, such as beaches and sandbars. Areas in close proximity to the nest are defended aggressively. Shallow water that is close by is also necessary (USGS 2001a). Least terns feed almost exclusively on small fish, but may also consume aquatic invertebrates (Gough 1998). Least terns dive for fish in water close to beaches or in quiet ponds and bays.

**Forster's tern (*Sterna forsteri*)**

It was not until 1831 that these terns were recognized as a species separate from the common tern. These medium-sized terns have a grey back and wings. They also have a black cap which turns white in the winter, leaving black marks behind the eyes. They have orange legs and an orange bill with a black tip. The sexes are similar in appearance and size. Forster's terns average 36 cm in length and have 76 cm wingspans. Their body weight ranges from 127 to 193 g with a mean of 158 g (SD=16.8 g; Dunning 1984). These birds range from their breeding grounds along the Atlantic coast from Massachusetts to Texas and, in the west, from Alberta to California, to their wintering grounds along the coasts of California and Virginia, and southward (Gough 1998; eNature Field Guide 2001). Forster's terns breed and summer in the Calcasieu region (Peterson 1980).

Forster's terns inhabit fresh and salt water marshes, lakes, streams, and beaches. In these environments, they feed on fish, crustaceans, aquatic insects, and mollusks. They often catch their prey by aerial diving. Forster's terns are colonial nesters. They have one brood each year, consisting of two to three buff, spotted eggs

(McLaren/Hart-Chemrisk 1998; Gough 1998; eNature Field Guide 2001). Forster's terns aggressively defend their nests and colonies from other bird species (Hebert 2000).

**Caspian tern (*Sterna caspia*)**

Caspian terns are large birds. Males and females are similar in size and appearance. Dunning (1984) reported a mean weight of 661 g (SD $\pm$ 37.6, N = 10). A 650-700 g range in weight was reported by Hebert (2000). The length from bill to tail tip is between 48 and 58 cm and wingspans are approximately 135 cm (Peterson 1980; Gough 1998). Caspian terns have large chests and long red beaks. They have grey backs and upper wings, a white underbelly, and a black cap on a white head (Gough 1998; Hebert 2000). Caspian terns are winter, nonbreeding inhabitants of the Calcasieu estuary (Peterson 1980).

Habitat preferences include open water environments like lakes, bays, wetlands, coastal waters, and beaches (Peterson 1980). Caspian terns primarily prey on fish, but will also eat aquatic invertebrates. Foraging occurs by hovering over the water and diving at fish near the surface (Gough 1998; Hebert 2000).

**Hypothetical Piscivorus Receptor**

The hypothetical piscivorus bird receptor was created by considering the life history and foraging data of the brown pelican, belted kingfisher, osprey, least tern, Forster's tern, and Caspian tern. This receptor is considered an average-sized piscivorus bird for the Calcasieu Estuary and possess the following qualities:

- The average-size receptor's weight is derived from the average of the six focal species. A 15% coefficient of variation (CV) for body weight is assumed. This value is a typical coefficient of variation found for wild

birds. A "what if" scenario will also be considered, where the receptor body weight will be set to the weight of the smallest bird (least tern) to consider species with the greatest potential for exposure (highest metabolic rate and food ingestion rate when normalized to body weight).

- The hypothetical receptor is assumed to have a relatively small foraging range with high site fidelity and no territoriality. BI AOC, MCR AOC, and UCR AOC regions of the Calcasieu Estuary system were identified as areas of concern for piscivorus birds in the deterministic risk assessment. It is assumed that receptors will forage exclusively within these areas.
- The receptor is a year round inhabitant in each of the Calcasieu Estuary areas. The temporal scale for this assessment is long term because: (1) COC levels are likely to exhibit low temporal variability because of their high persistence, and (2) chronic toxicity typically occurs at much lower levels than acute toxicity.
- It is assumed that the hypothetical receptor forages entirely on fish from classes 1, 2a, and 2b (<15 cm in length), and classes 3a and 3b (15 - <30 cm in length).

## 1.4 Conceptual Model

The conceptual model illustrates the relationships between sources and releases of COCs, their fate and transport, and the pathways through which COCs reach piscivorus birds and exert potential adverse effects. The model enhances the level of understanding regarding the relationships between human activities and ecological receptors at the site under consideration. In so doing, the conceptual model provides a framework for predicting effects on ecological receptors and a template for generating risk questions and testable hypotheses (USEPA 1997b; 1998). The

conceptual site model developed for the Calcasieu Estuary is described in greater detail in Chapter 7 of the BPF. It summarizes information on the sources and releases of COCs, the fate and transport of these contaminants, the pathways by which ecological receptors are exposed to the COCs, and the potential effects of these contaminants on the ecological receptors that occur in the Calcasieu Estuary. In turn, this information is used to develop a series of risk hypotheses that provide predictions regarding how ecological receptors will be exposed to and respond to the COCs.

Piscivorus birds are exposed to a number of COPCs in the Calcasieu Estuary system and the deterministic risk assessment (Appendix G) identified those COCs that pose potential risks to these animals. Specifically, piscivorus birds are at greatest risk from TCDD-TEQs, selenium, mercury, and total PCBs in the Calcasieu Estuary. These contaminants are persistent and bioaccumulative and are available for uptake by piscivorus birds, primarily through the food chain. The Phase II sampling program provided data identifying substantial tissue residues of these contaminants in fish, which are prey items for piscivorus birds. Other routes of exposure, including inhalation, water consumption and sediment ingestion have been excluded from this assessment as their contribution to overall exposure is likely negligible.

## 1.5 Assessment Endpoints

An assessment endpoint is an 'explicit expression of the environmental value that is to be protected' (USEPA 1997b). The selection of assessment endpoints is an essential element of the overall ERA process because it focuses assessment activities on the key environmental values (e.g., reproduction of piscivorus birds) that could be adversely affected by exposure to environmental contaminants. Assessment endpoints must be selected based on the ecosystems, communities, and species that

occur, have historically occurred, or could potentially occur at the site (USEPA 1997b).

Aquatic-dependent bird species are integrally linked to aquatic ecosystems as a result of their reliance on aquatic organisms for food. These species can be classified, based on their feeding habits, into three main groups: sediment probing birds (i.e., species that eat benthic macroinvertebrates), carnivorous wading birds (i.e., species that eat various types of aquatic organisms, including invertebrates, small fish, reptiles, amphibians) and piscivorus birds (i.e., species that eat fish). Due to their reliance on aquatic organisms for food, it is important to evaluate the effects of environmental contaminants on piscivorus birds.

The primary exposure pathway for piscivorus birds to COCs is through the ingestion of contaminated fish. Exposure is also possible through dermal contact with surface water and sediments, oral ingestion of water, and inhalation, but these routes are likely of minor importance. For example, piscivorus birds are unlikely to use the saline waters of BI AOC as a source of drinking water. Previous assessments have also demonstrated the inhalation route of exposure to be an insignificant source of methylmercury and PCBs to aquatic-dependent birds (e.g., Moore *et al.* 1999). Therefore, the exposure model used in this assessment only includes the ingestion of fish.

The assessment endpoint for this assessment is the survival, growth, and reproduction of piscivorus birds.

## 1.6 Measurement Endpoints

A measurement endpoint is defined as 'a measurable ecological characteristic that is related to the valued characteristic selected as the assessment endpoint' and it is a measure of biological effects (e.g., mortality, reproduction, growth; USEPA 1997b). Measurement endpoints are frequently numerical expressions of observations (e.g., toxicity test results, community diversity measures) that may or may not be compared to similar observations at a control and/or reference site.

Measurement endpoints for piscivorus birds were selected based on linkages between exposure media and receptors within the Calcasieu Estuary. This information was used to identify candidate measurement endpoints that could be used to evaluate the status of each assessment endpoint. Only the highest priority measurement endpoints were included in the RI Phase II sampling program. The measurement endpoints for piscivorus birds were the concentrations of bioaccumulative substances in the tissues of their prey species.

The potential for adverse effects on piscivorus birds will be evaluated using tissue chemistry data for prey species. Specifically, data on the concentrations of contaminants measured in small fish (i.e., <15 cm in length) and medium-sized fish (i.e., 15 to 30 cm) will be used. These data will be compiled by geographic area within the estuary (based on the diet and foraging range of each bird species) and compared to appropriate benchmark values for survival and reproduction of birds. In this evaluation, the tissue residue data for the fish collected in the estuary will be assumed to be similar to that for other fish species not captured during the sampling program.

To support the identification of key assessment and measurement endpoints for the Calcasieu Estuary BERA, the United States Environmental Protection Agency (USEPA) convened a BERA workshop in Lake Charles, LA on September 6 and 7, 2000. The workshop participants included representatives of the USEPA, United States Geological Service (USGS), National Oceanic and Atmospheric Administration (NOAA), Louisiana Department of Environmental Quality (LDEQ), United States Fish and Wildlife Service (USFWS) and CDM Federal. The workshop was designed to enable participants to articulate the goals and objectives for the ecosystem (i.e., based on the input that had been provided by the community in a series of public meetings), to assess the state of the knowledge base, to define key issues and concerns, and to identify the chemicals and areas of potential concern in the study area. This workshop provided a basis for refining the candidate assessment endpoints that had been proposed based on the results of the SERA (CDM 1999). Workshop participants also identified a suite of measurement endpoints that would provide the information needed for evaluating the status of the assessment endpoints (MacDonald *et al.* 2000).

## 1.7 Risk Hypothesis and Questions

The following risk hypothesis was developed to identify the key stressor-effect relationships that will be evaluated in the ecological risk assessment:

> Based on the physical-chemical properties (e.g., $K_{ow}$s) of the bioaccumulative contaminants of concern, the nature of the food web in the Calcasieu Estuary, and the effects that have been documented in laboratory studies, TCDD-TEQs, selenium, mercury, and total PCBs released into surface waters will

accumulate in the tissues of aquatic organisms to levels that adversely affect the survival, growth, and/or reproduction of piscivorus birds.

Assessment endpoints are linked to measurement endpoints via risk questions and testable hypotheses. The key risk questions for piscivorus birds include:

- Are the levels of COCs in the tissues of prey species of piscivorus birds in the Calcasieu Estuary higher than the benchmark values for survival, growth, or reproduction?
- If yes, what are the probabilities of effects of differing magnitude for survival, growth, and/or reproduction of piscivorus birds?

The linkages between the assessment endpoint and the measurement endpoints are articulated in greater detail in Table A1-21 of the Baseline Problem Formulation (MacDonald *et al.* 2001).

## 1.8 Purpose of the Appendix

The purpose of this assessment is to characterize the likelihood of piscivorus birds experiencing adverse effects associated with exposure to the COCs identified in Appendix G.

# 2.0 Methods

A step-wise approach was used to assess risks to the piscivorus bird community posed by the COCs in the Calcasieu Estuary. The steps in this process included:

- Collection, evaluation, and compilation of the relevant data on the concentrations of COCs in prey items in the Calcasieu Estuary;
- Assessment of exposure of piscivorus birds to COCs (Figure H3-1);
- Assessment of the effects of COCs on piscivorus birds (Figure H3-2); and,
- Characterization of risks to piscivorus birds (Figure H3-3).

Each of these steps is described in the following sections of this report. The results of the deterministic assessment were briefly reviewed in Section 1.1. For details of this assessment, see Appendix G.

## 2.1 Collection, Evaluation, and Compilation of Data

Information on contaminant levels in tissues of prey of piscivorus birds were collected in two phases, termed the Phase I and Phase II sampling programs. The Phase I program results indicated that the detection limits for many of the COCs in tissues were orders of magnitude above corresponding benchmarks. Therefore, the Phase I results for tissues were not considered in this assessment. The methods used to collect the tissue samples, quantify the levels of COCs, evaluate the reliability of the data, and compile the information in a form that would support the BERA are described in the following sections.

***Sample Collection of Tissues*** - More than 600 tissue samples were collected at sites located throughout the estuary between October, 2001 and November, 2001.

Biota tissue samples were collected in four AOCs in the estuary (upper, and mid Calcasieu, and Bayou d'Inde) and in the reference areas (Bayou Choupique, Grand Bayou, Bayou Bois Connine, Johnson Bayou, Willow Bayou). There were also a number of sub-areas within the AOCs from which samples were taken. The USEPA Region V FIELDS tools were used to randomly select coordinates (i.e., latitude and longitude) for the assigned number of primary sampling stations and alternate sampling stations (i.e., which were sampled when it was not possible to obtain samples from the primary sampling stations). In the field, each sampling station was located with the aid of navigation charts and a Trimble differentially-corrected global positioning system (GPS). Using standard statistical power analysis methods, an evaluation of previously collected data was completed to determine the number of samples to be collected within each area and sub-area.

The methods used to collect, handle, and transport the tissue samples are described in CDM (2000a; 2000b; 2000c; 2000d; 2000e). Briefly, fish species were collected by hook and line, and netting. Minnows and other small bait species were collected using legal cast nets, minnow traps, dip nets and bait seines in accordance with the Louisiana Department of Wildlife and Fisheries. Each sample was wrapped in aluminum and put in a Ziploc® bag. All samples were kept frozen and shipped to laboratories in coolers on dry ice.

***Chemical Analyses of Tissues*** - Chemical analysis of the tissue samples was conducted at various contract laboratory program (CLP) and subcontract (non-CLP) analytical laboratories, including USEPA Region VI Laboratory, USEPA Region VI CLP laboratories, Olin Contract laboratories, Texas A&M University laboratories,

ALTA laboratories, AATS laboratories and EnChem laboratories. Upon receipt at the laboratory, tissue samples were held in freezers until analysis.

All tissue samples were analyzed for total target analyte list (TAL) metals, target compound list (TCL) semi-volatile organic compounds (SVOCs) and TCL pesticides. Total metals were quantified using the SW6010B method. Polycyclic aromatic hydrocarbons and/or other semi-volatile organic compounds were quantified using the SW8270C method. Methods SW8081A and SW8082 were used to quantify pesticides. Twenty percent of the tissue samples were analyzed for PCB congeners and dioxins/furans. EPA Method SW1668 was used to quantify PCB congeners and SW8290 was used for dioxins/furans.

EnChem laboratories used additional analytical methods to quantify mercury, polycyclic aromatic hydrocarbons (PAHs), pesticides and dioxins and furans. Methods 1631MOD and 1630MOD were used to quantify mercury and methylmercury, respectively. PAHs were quantified using Method 8270C-SIM. Method SW8082 and AXYS Method CL-T-1668A/Ver.3 were used to quantify pesticides. Dioxins and furans were quantified using AXYS Method DX-T-8290/Ver.2.

***Data Validation and Verification*** - All of the data sets generated during the course of the study were critically reviewed to determine their applicability to the assessment of risks to the biotic community in the Calcasieu Estuary. The first step in this process involved validation of the tissue chemistry data. Following translation of these data into database format, the validated data were then further evaluated to ensure the quality of the data used in the risk assessment**.** We were unable to confirm tissue data results against the original source.

***Database Development*** - To support the compilation and subsequent analysis of the information on biota and sediment quality conditions in the Calcasieu Estuary, a relational project database was developed in MS Access format. All of the sediment, porewater and tissue chemistry, benthic community and sediment toxicity data compiled in the database were georeferenced to facilitate mapping and spatial analysis using geographic information system (GIS)-based applications (i.e., ESRI's ArcView and Spatial Analyst programs). The database structure made it possible to retrieve data in several ways, including by data type (i.e., chemistry vs. toxicity), by sediment horizon (i.e., surficial vs. sub-surface sediments), by stream reach (i.e., Upper Bayou d'Inde vs. Lower Bayou d'Inde), by sub-reach (i.e., Upper Bayou d'Inde-1 vs. Upper Bayou d'Inde-2), and by date (i.e., Phase I vs. Phase II). As such, the database facilitated a variety of data analyses.

## 2.2 Probabilistic Ecological Risk Assessment

Monte Carlo analysis is an increasingly widely used approach to probabilistic risk assessment (USEPA 1997c, 1999). It is used to propagate uncertainty associated with the variability of input variables, as well as incertitude associated with how to parameterize input distributions. In this assessment we use probability bounds analysis to determine the relative contributions of incertitude and variability to exposure estimates (see Chapter 9 of MacDonald *et al.* 2001 for more information on the uncertainty analysis approaches used here).

Monte Carlo analysis requires the specification of the statistical distributions of each of the input variables and their interdependencies as measured by correlations. Computer software such as Crystal Ball is used to 'sample' from these distributions and, via the exposure model equation, compute an exposure distribution. This

process is repeated many times so as to build up a histogram that serves as the estimate of the full distribution of exposures (explicitly including the tail risks of extreme exposure).

Probability bounds analysis is an exact numerical approach (not based on simulation) that takes as input the same probability distributions used in Monte Carlo simulation, or, when they are difficult to specify precisely, bounds on these distributions (Ferson *et al.* 2002). The method then rigorously computes bounds on the cumulative distribution function. The spread between the bounds of an input or output distribution corresponds directly to the amount of incertitude we have about how to describe the variable. Probability bounds analysis is also useful when independence assumptions are untenable (such as between concentrations in sediments and benthic invertebrates), or when sparse empirical data make it difficult to quantify the correlations among variables.

### 2.2.1 Exposure Characterization

The exposure model calculates the total daily intake of COCs associated with the ingestion of food. Chemical assimilation efficiency terms are not included in the exposure equation because the efficiencies of chemical adsorption in wild animals following ingestion will likely be similar to the efficiencies in laboratory animals in toxicity studies. Thus, the chemical assimilation efficiency terms will cancel out when the exposure and effect estimates are combined to estimate risk.

The temporal scale for this assessment is long term, because: (1) COC levels are unlikely to exhibit high temporal variability, and (2) chronic toxicity occurs at much lower levels than acute toxicity. The spatial scale of this assessment is considered to

be approximately equal to the foraging range typical of piscivorus birds (USEPA 1993, 1995). The foraging area for the hypothetical receptor is set to 40,000 $m^2$. This area is equivalent to a circular zone of 113 metres in diameter or 4,000 metres of shoreline 10-m wide, both of which easily fit into each sub-area of interest.

The exposure model is:

$$TDI = FMR \times \sum_{i=1}^{n} \frac{C_i}{AE_i \times GE_i} \qquad \textbf{EQUATION \#1}$$

where:

$TDI$ = total daily intake of contaminant (mg/kg bw/day),
$FMR$ = normalized free metabolic rate (Kcal/kg bw/day),
$C_i$ = concentration of contaminant in prey (mg/kg),
$GE_i$ = gross energy of prey (Kcal/kg prey),
$AE_i$ = assimilation efficiency of prey (unitless),

Each input variable is described in detail below, including the parameterizations for the Monte Carlo analyses and the probability bounds analyses.

#### 2.2.1.1 Selection Criteria for Input Distributions

The distributions and distribution parameters used in the exposure analyses are summarized in Tables H3-2 and H3-3. Input distributions were assigned as follows: lognormal distributions for variables that are positively skewed with a lower bound of zero and no upper bound (e.g., tissue concentrations), beta distributions for variables bounded by zero and one (e.g., prey assimilation efficiency), and normal distributions for variables that are symmetric and not bounded by one (e.g., body weight). The lognormal distribution is often used to provide good representations for

physical quantities constrained to being non-negative, and that are positively skewed, such as substance concentrations, stream flows, or magnitudes of accidents (Small 1990). Ott (1995) provides an extensive discussion of the theoretical reasons for why substance concentrations in the environment are expected to be lognormally distributed. The beta distribution provides a flexible means of representing variability over a fixed range, such as zero to one (Small 1990). The beta distribution can take on a wide variety of shapes between the fixed endpoints and this flexibility has led to its empirical use in diverse applications. The normal distribution arises in many cases because of the central limit theorem which results in a normal distribution for additive quantities such as body weights (Small 1990). The normal distribution can often be used for variables that are non negative, as long as coefficients of variation (CV) are small. This is because many distributions converge to a normal distribution as CVs become small. With most random number generators, it is impossible to obtain numbers more than five standard deviations from the mean. Thus, as long as the CV is less than 0.2, there is no concern for selecting negative values for non-negative variables.

#### 2.2.1.2 Input Distributions

***Body Weight (BW)***

Although body weight data are not used in the exposure model directly, they are a required variable in allometric models used to estimate the free metabolic rate. For this assessment, we used body weights that represent an average-sized and a small piscivorus bird.

For the Monte Carlo analysis, the average body weight of brown pelican, belted kingfisher, osprey, Caspian tern, Forster's tern, and least tern was used (i.e., 1.25 kg).

Because the feeding guild encompasses species with widely varying body weights, the calculation of the standard deviation of the mean body weight would have yielded an unduly wide distribution. Instead, we adopted a coefficient of variability (CV) of 15%, which is typical of body weight data for birds. The application of the adopted CV yielded a standard deviation of 0.188.

We also repeated the Monte Carlo analysis with a mean body weight of 0.0431 kg (standard deviation equal to 0.00647). This body weight is representative of the smallest bird in the guild, the least tern. Small birds tend to have higher metabolic rates and, as a result, may be at higher risk of exposure.

Body weights were assumed to be distributed normally. The entire proportion of uncertainty in this variable is likely due to variability, with little incertitude. Thus, probability bounds were not established for this input variable.

***Free Metabolic Rate (FMR)***

To estimate free metabolic rate, the allometric equation derived by Nagy (1987) was used:

$$FMR = a \cdot BW(g)^b \qquad \textbf{EQUATION \#2}$$

A probabilistic approach was used to estimate FMR in both the Monte Carlo and probability bounds analyses, wherein distributions were derived for each of the input variables (body weight [*BW*, *a*, *b*]) and combined according to the above equation. The slope (*a*) and power term (*b*) distributions were based on the error statistics reported in Nagy (1987), assuming an underlying normal distribution for each. For piscivorus birds, log *a* had a reported mean of 0.681 and a standard error of 0.102, and *b* had a reported mean of 0.749 and a standard error of 0.037 (Nagy 1987). The body weight (*BW*) distribution was described above.

### *Gross Energy of Invertebrates ($GE_i$)*

Gross energies of fish were available from the literature. The mean gross energy of fish used in the exposure model and probability bounds was 1200 Kcal/kg (standard deviation = 240, Thayer *et al.* 1973). The distribution for this variable was assumed to be lognormal. Incertitude was considered low for this input variable because: (1) sufficient experimental data were available to confidently estimate the mean and standard deviation, (2) the variable is easily measured and thus measurement error is low, and (3) there appears to be little difference in the gross energies of different prey species. Therefore, probability bounds were not derived for this variable.

### *Assimilation Efficiency ($AE_i$)*

A mean assimilation efficiency of 79% (standard deviation = 4.5) was used for piscivorus birds. This assimilation efficiency was reported by Karasov (1990), Stalmaster and Gessaman (1982), Castro *et al.* (1989) and Ricklefs (1974). In the Monte Carlo analyses, a beta distribution was assumed for this variable with the following parameterization: alpha = 15, beta = 4, and scale = 1. Incertitude was considered low for this input variable because: (1) the variable is easily measured and thus measurement error is low, and (2) there appears to be little difference in the gross energies of different prey species. Therefore, probability bounds were not derived for this variable.

### *Concentration of COCs in fish ($C_i$)*

Concentrations of COCs were obtained using tissue chemistry data from fish species collected in each AOC. Concentrations represent the level of COCs in fish from classes 1, 2a and 2b (< 15 cm in length), 3a and 3b (15 - < 30 cm in length). Fish species collected included, but were not limited to, gulf killifish, gulf menhaden, spot, Atlantic croaker, and spotted seatrout. Mean concentrations were derived by fitting the data to a lognormal distribution using Crystal Ball 2000 (Decisioneering 2000).

Piscivorus birds are likely to spatially and temporally average their exposure within each region over the long term. Mean tissue concentrations used in the Monte Carlo analyses were derived using bootstrapping, based on average daily exposure over 160 foraging days. Mean tissue concentrations used to calculate probability bounds were derived using the Land statistic. The means and standard deviations calculated using these methods are provided below for each COC and corresponding AOC. Unless noted otherwise, all concentrations are in wet weight.

*TCDD-TEQs*

The fitted means for TCDD-TEQs in fish tissue were 82.6 ng/kg (SD = 68.0, n = 33) from BI AOC, 31.3 ng/kg (SD = 42.1, n = 8) from MCR AOC, and 25.4 ng/kg (SD = 22.3, n = 6) from the reference areas. The bootstrapped grand means and standard deviations were 82.7 ng/kg and 2.06 for BI AOC, 32.0 ng/kg and 1.63 for MCR AOC, and 25.3 ng/kg and 0.640 for the reference areas. Using the Land statistic, the means and standard deviations were 93.1 ng/kg and 15.8 for BI AOC, 59.5 ng/kg and 44.6 for MCR AOC, and 58.0 ng/kg and 44.6 for the reference areas.

*Selenium*

Concentrations of selenium in fish tissue from BI AOC had a fitted mean of 0.554 mg/kg (SD = 0.187, n = 138). Fish tissue levels from the reference areas had a fitted mean of 0.530 (SD = 0.415, n = 45). The bootstrapped grand means and standard deviations were 0.552 mg/kg and 0.00587 for BI AOC, and 0.530 mg/kg and 0.0130 for the reference areas. Using the Land statistic, the means and standard deviations were 0.583 mg/kg and 0.0346 for BI AOC, and 0.574 mg/kg and 0.0857 for the reference areas.

*Mercury*

The fitted means for mercury in fish tissue were 0.173 mg/kg (SD = 0.151, n = 138) from BI AOC, 0.507 mg/kg (SD = 0.0378, n = 83) from UCR AOC, 0.0558 mg/kg (SD = 0.0255, n = 58) from MCR AOC, and 0.0258 mg/kg (SD = 0.0136, n = 47) from the reference areas. The bootstrapped grand means and standard deviations were 0.174 mg/kg and 0.00469 for BI AOC, 0.0507 mg/kg and 0.00117 for UCR AOC, 0.0556 mg/kg and 0.000944 for MCR AOC, and 0.0259 mg/kg and 0.000408 for the reference areas.

Using the Land statistic, the means and standard deviations were 0.179 mg/kg and 0.0154 for BI AOC, 0.0537 mg/kg and 0.00576 for UCR AOC, 0.0605 mg/kg and 0.00625 for MCR AOC, and 0.0282 mg/kg and 0.00345 for the reference areas.

*Total PCBs*

Concentrations of total PCBs in fish tissue from BI AOC had a fitted mean of 0.187 mg/kg (SD = 0.466, n = 138). Fish tissue levels from the reference areas had a fitted mean of 0.0269 (SD = 0.0581, n = 46). The bootstrapped grand means and standard deviations were 0.184 mg/kg and 0.0115 for BI AOC, and 0.0265 mg/kg and 0.00181 for the reference areas. Using the Land statistic, the means and standard deviations were 0.186 mg/kg and 0.0271 for BI AOC, and 0.0284 mg/kg and 0.00700 for the reference areas.

### 2.2.1.3 Monte Carlo Analyses

The Monte Carlo analyses for exposure combined the input distributions as specified in Section 2.2.1. Each analysis included 10,000 trials and Latin Hypercube Sampling to ensure adequate sampling from all portions of the input distributions. The analyses

were done in Crystal Ball 2000 (Decisioneering 2000). Considering all possible pairwise combinations of input variables (Table H3-2), no dependencies were expected except for concentrations of COCs in fish. Therefore, no correlations were included in the Monte Carlo analyses. The Monte Carlo analyses made no distinction in the way incertitude and variability were propagated; they were simply combined. We address incertitude and variability separately in the probability bounds analyses described below.

#### 2.2.1.4 Probability Bounds Analyses

The probability bounds analyses were run using RiskCalc, version 4.0 (Ferson 2002). The input variables used in the probability bounds analysis are presented in Table H3-3. With the exception of tissue concentrations, the input variables are similar to those used in the Monte Carlo analyses. Mean tissue concentrations used in the probability bounds analyses were calculated using the Land statistic.

### 2.2.2 Effects Characterization

The purpose of this section is to: (1) briefly review the literature on the effects of TCDD-TEQs, selenium, mercury, and total PCBs to piscivorus birds, and (2) select the appropriate effects metric for each COC to be used with the results of the exposure assessment to estimate risk. We will focus on ecologically relevant effects endpoints such as survival, reproduction, and growth. Examples of piscivorus bird species considered in this section include brown pelicans, belted kingfishers, osprey, and terns. Because the available toxicological information for these species is limited, data from other bird studies will be discussed where appropriate. Other

information on the toxicity of these COCs to wildlife can be found in Appendices 5 and 10 of the problem formulation document (MacDonald *et al.* 2001).

Effects data can be characterized and summarized in a variety of ways ranging from benchmarks designed to be protective of most or all species to dose-response curves for the receptor group of interest (i.e., piscivorus birds). In this assessment, effects characterization preferentially relies on dose-response curves, but defaults to benchmarks or other estimates of effect (e.g., no observed adverse effect level (NOAEL), lowest observed adverse effect level (LOAEL) when insufficient data are available to derive dose-response curves. Effects associated with growth, survival, and reproduction are generally the preferred measures of effect.

The following is the hierarchy of decision criteria used to characterize effects for each receptor group-COC combination:

1. Had bioassays with five or more treatments been conducted on the receptor group of interest or a reasonable surrogate? If yes, the dose-response relationship was estimated using the Generalized Linear Model (GLiM) framework described in Kerr and Meador (1996) and Bailer and Oris (1997). The GLiM framework involves conducting linear regression analysis on dose-response data that have been transformed to linearize the relationship (e.g., probit transformation for survival data). If not, we proceeded to 2.

2. Were multiple bioassays available that, when combined, had five or more treatments on the receptor group of interest or a reasonable surrogate? Such bioassays would be expected to have had similar protocols, exposure

scenarios and effects metrics. If yes, we estimated the dose-response relationship as in 1. If not, we proceeded to 3.

3. Had bioassays with less than five treatments been conducted on the receptor group of interest or a reasonable surrogate? If yes, we conducted hypothesis testing to determine the NOAEL and LOAEL or reported these metrics when available from the original study. If not, we proceeded to 4.

4. Were sufficient data available from field studies and monitoring programs to estimate concentrations or doses of COCs consistently associated with no adverse effects and with adverse effects to piscivorus birds? If yes, we developed field-based no effects and effects measures. This approach is analogous to the approach used to develop sediment-quality guidelines for the protection of aquatic life (see Long *et al.* 1995; MacDonald *et al.* 1996; MacDonald *et al.* 2000). If not, we proceeded to 5.

5. We derived a range within which the threshold for the receptor group of interest was expected to occur. Because information on the sensitivity of the receptor of interest was lacking, it was difficult to derive a threshold that was neither biased high or low. If bioassay data were available for several other species, however, one could calculate a threshold for each to determine a threshold range that spanned sensitive and tolerant species. That range was likely to include the threshold for the receptor group of interest.

### 2.2.2.1 TCDD-TEQs

This section will examine the effects of TCDD (2,3,7,8-tetrachlorodibenzo-*p*-dioxin) and equivalents to piscivorus birds. The TCDD Equivalent (TEQ) approach relates the toxicity of specific PCB (polychlorinated biphenyl), PCDD (polychlorinated dibenzo-*p*-dioxin), and PCDF (polychlorinated dibenzofuran) congeners to that of TCDD. This technique provides a basis with which to compare the results of toxicity studies involving PCB, PCDD, and PCDF mixtures and congeners to the specific congener profiles of sites in the Calcasieu Estuary system and is described further in Appendix G. Literature relating to survival, growth, and reproduction was reviewed. The focal species in this section are belted kingfishers, osprey, terns, and brown pelicans. Additional species will be included in the discussion when necessary. Concentration data are in wet weight (ww), unless noted otherwise.

***Survival***

Nosek *et al.* (1992a) treated mature hen pheasants with single intraperitoneal TCDD injections of 6,250, 25,000, or 100,000 ng/kg bw. These birds suffered body weight loss and mortality at the two higher dose levels. All birds given the 100,000 ng/kg bw dose were dead by the sixth week and 75% of those given 25,000 ng/kg bw were dead by the twelfth week. These investigators also examined the subchronic effects of TCDD to pheasants, dosing birds weekly with 10, 100, or 1,000 ng/kg bw for ten weeks (cumulative doses of 100, 1,000, or 10,000 ng/kg bw). Fifty-seven percent of birds given the highest dose died within the 24 week experiment, while those on the lower doses experienced no mortality. Bobwhite quail, mallards and ringed turtledoves given single oral doses of TCDD were found to have 37-day $LD_{50}$s of 15,000, 108,000, and 810,000 ng/kg bw, respectively (Hudson *et al.* 1984). Chickens given single oral doses of 25,000 ng/kg died within 12 days (Grieg *et al.* 1973) and

a 21 day oral NOAEL of 100 ng/kg/day was reported for treatments to 3 day old white leghorn chicks (Schwetz *et al.* 1973).

***Reproduction***

Nosek *et al.* (1992a) monitored egg production and embryonic mortality in mature hen ring-necked pheasants after weekly intraperitoneal injections of 10, 100, and 1,000 ng/kg bw TCDD. At the highest dose, egg production fell significantly compared to controls - from a cumulative total of 33 eggs per bird down to 12. Egg production was not affected at the two lower doses. Embryotoxicity significantly increased in response to the dose level. Cumulative doses of 100 and 1,000 ng/kg bw elicited insignificant increases in embryo mortality, but the 10,000 ng/kg bw dose caused 100% embryo mortality compared to 0% in controls.

The effects of egg injection of TCDD , TCDF, and PCBs have been reported by several investigators and include decreased egg production and increased embryonic mortality. Studies of egg injections with PCBs have demonstrated that when similar toxicant levels are attained in the egg via injection and via conventional maternal dietary doses, the effects to the chicks are also similar (Hoffman *et al.* 1996a; Nosek *et al.* 1993). Embryonic uptake of organochlorines from yolk is similar for substances injected into the yolk and for those accumulated naturally (Peakall and Fox 1987). Bioaccumulative environmental substances concentrate in egg yolks (Tumasonis *et al.* 1973; Custer *et al.* 1997). As a result, many studies have been conducted examining the effects of injecting environmentally relevant concentrations of PCBs into yolks. Egg yolk injected PCBs are distributed throughout the embryo, including fat tissue, liver, kidneys and bone marrow (Brunstrom *et al.* 1982). Ring-necked pheasant hens fed radiolabeled TCDD were found to eliminate approximately 1% of their body burdens into eggs, and all of the substance was deposited in the yolk, none in the albumin (Nosek *et al.* 1992b). The maternal transfer of total PCBs to eggs for

several avian species was investigated by Drouillard and Norstrom (2001). Ratios of egg yolk to maternal adipose tissue PCB concentrations ranged from 0.270 in ring doves to 1.20 in chickens and pheasant.

Henshel *et al.* (1997) estimated the $LD_{50}$ of TCDD injected into white leghorn chicken eggs yolks to be 122 ng/kg egg (by probit analysis, 146 ng/kg egg when determined by interpolation) and Powell *et al.* (1996b) observed that hatchability of white leghorn chicken eggs significantly decreased at a dose of 160 ng/kg egg TCDD injected into egg yolks. McKinney *et al.* (1976) reported that the injection of 5,000 ng/kg ww egg 2,3,7,8-TCDF (500 ng/kg ww egg TEQ) resulted in complete mortality of one-day-old white leghorn chickens within an average of 11.5 days. Chickens fed diets containing fish from a TCDD and PCB contaminated site at increasing concentrations experienced time and dose related decreases in egg hatchability (Summer *et al.* 1996). Total PCB concentrations in the diet ranged from 0.300 to 6.60 mg/kg. This corresponds to concentrations of 3.3 to 59 ng/kg diet of TCDD (TEQ), determined by the H4IIE bioassay.

Cormorant eggs were less sensitive to the effects of TCDD. Eggs collected from an isolated colony in Manitoba were injected with 4,000 ng/kg TCDD into the yolk sac. Cormorant eggs receiving 4,000 ng/kg egg TCDD suffered 50% mortality while controls experienced 28% mortality (Powell *et al.* 1997) in one experiment. The investigators then increased the dose range in a subsequent study (Powell *et al.* 1998) and observed 44.7% mortality in controls and 84.9% in eggs treated with 11,900 ng/kg egg TCDD. The $LD_{50}$ of this second study was estimated to be 4,000 ng/kg egg.

Nosek *et al.* (1993) estimated a TCDD $LD_{50}$ of 2,180 ng/kg ww egg to ring-necked pheasants when administered in egg yolks. Eggs were injected on day 0 of embryonic

development with doses of 10, 100, 1,000, or 10,000 ng/kg egg and mortality, defined as a "failure of the hatchling to emerge completely from the shell alive", was monitored. Eggs treated with the three lowest doses showed no significant increase in embryo mortality over controls, while the 10,000 ng/kg dose caused near total (98%) embryonic failure.

Chicken eggs that had been incubating for four days were injected with 3,3',4,4',5-pentachlorobiphenyl (PCB126) at treatment levels ranging from 0 to 2,000 ng/kg (0 to 200 ng/kg WHO TEQ; Brunstrom and Andersson 1988). After 14 days, embryonic mortality was highest in the highest treatment group (90%) compared to control groups (vehicle only = 15% embryo mortality). Brunstrom (1989) found that PCB126 was the most toxic of the congeners tested, with PCB77, 105, and 118 being 5, 1,000, and 8,000 times less toxic, respectively.

Powell *et al.* (1996a; 1996b; 1997) also investigated the embryotoxicity of PCB126 to chickens, and cormorants. Chicken eggs were yolk-injected with 100,000, 200,000, 400,000, 800,000, 1,600,000, 3,200,000, 6,400,000, and 12,800,000 ng/kg egg prior to incubation. The $LD_{50}$ for chick embryos was estimated to be 2,300 ng/kg egg (230 ng/kg egg TEQ). Cormorant eggs were collected from Lake Winnipegosis in Manitoba, Canada and were injected with doses of PCB126 at levels of 0, 5,000, 10,000, 25,000, 50,000, 100,000, 200,000, 400,000, and 880,000 ng/kg ww egg (0 to 88,000 ng/kg ww egg TEQ). The eggs were then incubated for 21 days and candled on days 7, 14, and 21 to check for viability. Significant increases in embryo mortality were observed in the 400,000 and 880,000 ng/kg dose groups (40,000 and 88,000 ng/kg TEQ), to 87% and 100%, respectively (Powell *et al.* 1997). An $LD_{50}$ of 158,000 ng/kg (15,800 ng/kg TEQ) was estimated. A second study involving cormorants estimated a PCB126 $LD_{50}$ of 177,000 ng/kg ww egg (17,700 ng/kg ww egg TEQ) after a single injection into the yolk (Powell *et al.* 1998).

PCB77 is another PCB congener whose toxicity closely resembles that of TCDD. Chicken eggs injected into the yolk with 5,000 or 20,000 ng/kg egg (250 or 1,000 ng/kg egg TEQ) showed significantly higher embryonic mortality (55 and 100%) than in controls (15%). Herring gull and goose eggs injected with doses as high as 1,000,000 ng/kg egg (50,000 ng/kg egg TEQ) showed no significant increases in mortality and duck eggs showed no significant increases in mortality with doses as high as 5,000,000 ng/kg egg (250,000 ng/kg egg TEQ; Brunstrom 1988). Wild turkey embryos were also much less sensitive to PCB77 than were chickens. The Ah receptor, thought to be instrumental in the expression of TCDD and PCB toxicity, is not present in turkeys in the embryonic stage of development, and may therefore provide a basis for the species difference (Brunstrom and Lund 1988).

Henshel *et al.*(1997) compared the relative sensitivities of TCDD yolk and air sac injections into the eggs of white leghorn chickens. Eggs were injected on day 0 of embryonic development and were allowed to hatch undisturbed. The result was a significantly (60%) lower $LD_{50}$ for the yolk route of administration. Air sac injections of PCB126 were also investigated by Hoffman *et al.* (1998) in multiple bird species. White leghorn chicken embryo was the most sensitive with an $LD_{50}$ of 400 ng/kg ww egg (40 ng/kg ww egg TEQ), while American kestrel and common tern embryos were less sensitive with $LD_{50}$s of 65,000 and 104,000 ng/kg ww egg (6,500 and 10,400 ng/kg ww egg TEQ), respectively. An $LD_{50}$ of 8,600 ng/kg egg (430 ng/kg TEQ) was calculated for chick eggs dosed with PCB77 administered into the air sac (Brunstrom and Andersson 1988).

***Other Effects***

Henshel (1998) dosed white leghorn chicken embryos with TCDD via yolk injection and examined the symmetry of the tectum and forebrain of the chicks' brains. Chickens suffered brain deformities, as asymmetries, at doses as low as 10 ng

TCDD/kg egg administered via egg yolk injection. Herons and cormorants showed brain asymmetry at accumulated TCDD levels of 10 and 19 ng/kg egg. Investigations of the teratogenic effects of PCB126 in chicks revealed the potential for beak deformities and edema (Powell *et al.* 1996a; 1996b). Injections of PCB126 at levels of 900 ng/kg egg (90 ng/kg egg TEQ) caused a significant increase in the number of abnormal embryos per number of eggs (13/60 vs 3/59 for the vehicle control) while having no significant impact on mortality of the birds. Other abnormalities noted included small or missing eyes and curved toes.

Weight gain of chicks is also an effect of PCB and TCDD exposure. White leghorn cockerels were fed a variety of hexachlorobiphenyl congeners at 400,000,000 ng/kg diet for 21 days and body weights monitored (McKinney *et al.* 1976). Three of five congeners tested significantly inhibited weight gain of the birds, with 2,4,5,2',4',5'-HCB having the most impact (chick weight 78% of controls on day 21). One congener, 3,4,5,3',4',5'-HCB, produced 100% mortality in test animals within 11 days of the onset of the experiment. Nestling kestrels orally dosed with PCB126 to levels of 50,000, 250,000, and 1,000,000 ng/kg bw/day (5,000, 25,000 and 100,000 ng/kg bw/day TEQ) via the diet also experienced inhibited weight gain (Hoffman *et al.* 1996b). For days 4 to 10 of the study, there was a significant correlation between PCB concentration and decreased body weight. Smaller bone lengths also indicated a reduced growth rate. Humerus, radius-ulna, and tibiotarsus were all significantly shorter in the 250,000 and 1,000,000 ng/kg bw/day (25,000 and 100,000 ng/kg bw/day TEQ) test groups than controls. Embryonic exposure to PCB126 also resulted in decreased growth rates in white leghorn chickens (Powell *et al.* 1996a). Injection of 900 ng/kg egg (90 ng/kg egg TEQ) of PCB126 prior to incubation produced significantly reduced body weights by the second week and 3,000 ng/kg egg (150 ng/kg egg TEQ) of PCB77 reduced body weights compared to controls at 3 weeks (Powell *et al.* 1996a).

### *Field Studies*

There has been some discussion in the literature regarding the relationship between adverse reproductive effects to birds observed in the field and long-lived chlorinated organic pollutants (de Voogt *et al.* 2001). PCBs and DDE are ubiquitous pollutants found at many contaminated sites. Cormorants in the Great Lakes area have a strong correlation ($r^2$ = 0.703) between egg mortality and bioassay-derived dioxin equivalents (Tillitt *et al.* 1992). Custer *et al.* (1999) instead suggest that DDE was primarily responsible for the observations of *in situ* decline in cormorant reproductive success in this area and that TCDD equivalents did not have a significant effect on cormorant reproductive success in Green Bay, despite significant PCB contamination. Eggs containing 299 ng TEQ/kg egg had 39% mortality while eggs from the reference site, containing only 35 ng TEQ/kg egg had 8% mortality. DDE concentrations were not included in the analyses.

Elliott *et al.* (2001) investigated the effects of organochlorine substances on the reproductive success of osprey in the Fraser and Columbia river systems. Analysis of concentrations in egg yolks and the results of laboratory incubation of eggs from the test and reference sites showed no correlation between embryonic mortality and *in ovo* substance exposure, despite hatching success ranging from 56 to 100% at the various sites. Woodford *et al.* (1998) monitored reproductive success of osprey exposed to chlorinated substances in the Wisconsin River from 1992 to 1996. Study sites included two test sites downstream of two bleached-kraft facilities and two reference sites upstream. From these sites, eggs were collected to measure contamination levels and the remaining eggs monitored for hatching and fledging rates as well as weight gain. Exposure to PCDDs, PCDFs and coplanar PCBs at these sites did not affect hatching or fledging rates, but chick growth may have been reduced at TCDD concentrations ranging from 54 to 67 ng/kg ww egg.

PCBs and dioxins have been linked to teratogenic effects in the field. Ludwig *et al.* (1996) observed a relationship between the abnormality rate (number of abnormalities per 1000 eggs) and TEQ concentrations in double-crested cormorants and Caspian terns in the upper Great Lakes. Bill defects and edema were the most common deformities. The abnormality rate reached 14.3% in live cormorant eggs in Green Bay, WI and 28.8% in live tern eggs in Saginaw Bay, MI, both contaminated sites. The overall live cormorant egg deformity rate correlated positively with TEQs ($r^2$ = 0.86) and the overall live tern egg deformity rate did not correlate as well with TEQs ($r^2$ = 0.12).

***Effects Metrics***

The most sensitive responses in birds exposed to TCDD-TEQs are associated with reproduction following long-term exposure. None of the available reproduction studies included species that could be considered piscivorus birds or reasonable surrogates. The hypothetical receptor embodies characteristics similar to brown pelicans, osprey, belted kingfishers, and terns. The lack of toxicity data for piscivorus birds exposed to TCDD-TEQs precludes the development of a dose-response curve (using either a single or multiple studies) or the derivation of a NOAEL and LOAEL for piscivorus birds (i.e., Options 1-3 in the hierarchy of decision criteria for choosing effects metrics are unavailable). The field data were insufficient to develop field-based benchmarks for piscivorus birds of the Calcasieu Estuary, which eliminates Option 4 for choosing effects metrics.

The final option for choosing an effects metric for piscivorus birds exposed to TCDD-TEQs is to derive a range within which the threshold for this receptor group is expected to occur. The most sensitive response observed was reproductive success of ring-necked pheasants injected weekly with TCDD (Nosek *et al.* 1992a). In this study, 14 ng TCDD/kg bw/day did not significantly reduce reproductive success of

hen pheasants, while the next highest dose of 140 ng TCDD/kg bw/day caused a decrease in cumulative egg production. This concentration will be used to represent the sensitive end of the toxicity threshold.

The upper bound of the threshold range is derived from a study on the effects of PCB126 to American kestrel hatchlings (Hoffman 1996a). In this study, the highest level of TEQ in the diet that did not cause adverse effects was 5,000 ng/kg TEQ. This dietary concentration is multiplied by the food intake rate of American kestrel hatchlings (0.00778 kg/day, Nagy 1987) and normalized to their body weight (0.076 kg, Hoffman *et al.* 1996b) to derive the corresponding dose:

$$UT = \left( \frac{5{,}000\ ng\ TEQ}{kg\ diet} \times \frac{7.78\ g\ food}{day} \times \frac{1.00\ kg}{1000\ g} \right) / 0.0760\ kg\ BW \quad \textbf{EQUATION \#3}$$
$$= 512\ ng / kg\,bw / day$$

where *UT* is the upper threshold dose and *BW* is body weight. Therefore, the threshold range for piscivorus birds exposed to TCDD-TEQs is 14 to 512 ng TEQ/kg bw/day.

### 2.2.2.2 Selenium

Selenium is an element required by birds and wildlife for good health. However, the range in concentration from healthy to toxic levels is very narrow (Heinz 1996). In nature, birds are exposed to different forms of selenium, each with varying degrees of accumulation and toxicity. Inorganic forms of selenium, such as selenite and selenate, are toxic to birds, but not to the same extent as organic selenides. Of these, selenomethionine is considered to be the most toxic and the most likely to harm birds

(Heinz 1996). Symptoms of selenium toxicity (selenosis) include decreased body weight and emaciation, hepatotoxicity, histologic lesions, and reproductive effects (Heinz 1996; Eisler 2000). The toxicity of different forms of selenium to birds has received little attention in laboratory studies and there is still much to be learned. Concentration data are in wet weight (ww), unless noted otherwise.

***Survival***

Laboratory studies have found that concentrations of selenium in the diet equal to or below 5 mg/kg are not associated with a decrease in bird health or survival. Mallards fed seleno-DL-methionine for 14 weeks at concentrations of 1, 2, or 4 mg/kg did not exhibit physiological effects of selenosis. In fact, mallards in the 2 and 4 mg/kg treatment groups gained weight (about 10%) compared to the controls (Hoffman *et al.* 1991). American kestrels (*Falco sparverius*) similarly did not show signs of selenosis when fed a diet containing 5 mg/kg dry weight (dw) of seleno-L-methionine for 77 days (Yamamoto *et al.* 1998).

The effects of dietary concentrations above 5 mg/kg dw of selenium vary. Hoffman *et al.* (1991) reported effects associated with a diet of 8 mg/kg of seleno-DL-methionine in adult male mallards. In this 14-week study, mallards in the 8 mg/kg group exhibited symptoms of hepatotoxicity, including responses to hepatic oxidized glutathione (GSSG) concentrations and the ratio of GSSG to reduced glutathione (GSH). These findings differed from Yamamoto *et al.* (1998), who found no observable symptoms of selenosis in male and female American kestrels fed 9 mg/kg dw of seleno-L-methionine.

Adult male and female mallards fed diets containing 13.3 mg/kg of seleno-DL-methionine for 150 days did not have body weights significantly different from controls and showed no signs of selenosis (O'Toole and Raisbeck 1997). These

findings were similar to those of Green and Albers (1997) for adult male mallards given 10 mg/kg of seleno-DL-methionine in the diet for 16 weeks. No histologic lesions, fat or muscle changes were identified in euthanized mallards. Heinz *et al.* (1988) described the dietary concentration of 10 mg/kg as being close to a no effects level for gross effects such as decreased food consumption, growth and survival. Their study used ducklings hatched from uncontaminated eggs and tested the effects of two types of selenium. One group of ducklings was fed a diet with 10 mg/kg of sodium selenite. Ducklings in this group had enlarged livers. The authors believed this could be attributed to the stimulation of detoxifying agents in the liver. Statistically significant reductions in food consumption and growth were also noted during the fourth and second weeks of the study, respectively. The second group of ducklings was fed 10 mg/kg dw of selenomethionine and displayed no significant effects (Heinz *et al.* 1988).

As dietary concentrations exceed those discussed above, the adverse effects of selenium toxicity become increasingly apparent and harmful. American kestrels fed a dietary concentration of 12 mg/kg seleno-DL-methionine had higher lean mass compared to total body weight and lower normalized body fat than controls or birds fed 6 mg/kg (Yamamoto and Santolo 2000). This raises concerns that wild kestrels exposed to these concentrations will be in poorer condition for the rigors of overwintering, migration, and breeding (Yamamoto and Santolo 2000).

Elevated liver and plasma GSH peroxidase was reported in mallard ducklings consuming 15 mg/kg of either seleno-L-methionine, seleno-DL-methionine, selenized yeast, or high-selenium wheat (Hoffman *et al.* 1996c). These symptoms are associated with hepatotoxicity in birds (Hoffman *et al.* 1991). This study found that seleno-L-methionine was more toxic (caused greater mortality) than seleno-DL-methionine under certain conditions. This finding is important because seleno-DL-

methionine is the organic form of selenium most often used in toxicity studies. Therefore, it is possible, under some conditions, that the toxicity of selenium existing as seleno-L-methionine may be underestimated (Hoffman *et al.* 1996c). A dietary concentration of 16 mg/kg of seleno-DL-methionine, administered to adult male mallards over 14 weeks, resulted in decreased body weights and lower hemoglobin concentrations compared to controls (Hoffman *et al.* 1991).

Ducklings fed dietary treatments containing either sodium selenite or selenomethionine at 20 mg/kg consumed less food than lower dose groups. As a result, growth rates for these mallards decreased (Heinz *et al.* 1988). Lower body weights than controls was also reported by O'Toole and Raisbeck (1997) for mallards consuming 25 mg/kg of seleno-DL-methionine. Hoffman *et al.* (1996c) examined the effects of different types of selenium (seleno-L-methionine, seleno-DL-methionine, selenized yeast, and high-selenium wheat) on ducklings at dietary concentrations of 15 and 30 mg/kg. The group receiving 30 mg/kg seleno-L-methionine yielded the lowest survival rate (36%) for the 2 weeks of exposure. All ducklings had elevated levels of plasma GSH peroxidase and lowered hematocrit concentrations (Hoffman *et al.* 1996c). Thirty-two mg/kg of seleno-DL-methionine resulted in weight loss, decreased hemoglobin and hematocrit concentrations, and histopathological effects in the liver (Hoffman *et al.* 1991).

Heinz *et al.* (1988) performed a 6 week study on mallard ducklings comparing the effects of sodium selenite and selenomethionine. A dietary concentration of 40 mg/kg of sodium selenite resulted in 25% mortality. The same dietary concentration as selenomethionine resulted in 12.5% mortality. At 80 mg/kg of sodium selenite, food consumption and body weight decreased after the first week. Ninety-eight percent mortality of ducklings was observed in this group over the 6 week study. One hundred percent mortality was recorded for ducklings consuming 80 mg/kg as

selenomethionine (Heinz *et al.* 1988). Heinz *et al.* (1988) observed that as the treatment level of selenium increased, so too did the amount of selenium in the liver of birds. Further, as the concentration of selenium in the liver increased, body weight decreased. Other studies have reported similar results for treatments above 30 mg/kg selenium (Heinz *et al.* 1987; Heinz *et al.* 1988; Green and Albers 1997; O'Toole and Raisbeck 1997). Albers *et al.* (1996) reported results similar to those listed above in their 16 week study of mallards exposed to either 0, 10, 20, 40, or 80 mg/kg seleno-DL-methionine. Symptoms associated with fatality observed in the 40 and 80 mg/kg groups included low body weight (25-50% below normal), emaciation, atrophy of fat and breast muscles, and liver necrosis. Symptoms observed in survivors included low body weight (10-15% below normal), poor plumage, reduced hatching success, and lipid peroxidation (Albers *et al.* 1996).

No adverse effects to survival were reported in the literature below a dietary concentration of 5 mg/kg of selenium (most often as selenomethionine). A dietary concentration of 10 mg/kg appears to be close to a no observed effects levels (Heinz *et al.* 1988). Adverse effects become more prominent as dietary concentrations exceed 10 mg/kg. Symptoms include loss of body weight, emaciation, histologic lesions, and hepatotoxicity. These symptoms become increasingly severe and harmful as dietary concentration increases, ultimately leading to death.

***Reproduction***

The embryo is the most sensitive avian life stage to selenium poisoning (Heinz *et al.* 1987; Hoffman and Heinz 1988; Heinz *et al.* 1989; Heinz 1996). Selenium levels in the egg provide the most sensitive measure for evaluating the potential for selenium toxicity. Effects to reproduction include embryo abnormalities, reduced hatchability, teratogenic effects, and reduced survival.

Reproductive effects occur at dietary concentrations below the threshold of adverse effects in adult birds. Heinz *et al.* (1987) studied reproductive effects from selenium using adult mallards fed either 1, 5, 10, 25, or 100 mg/kg of sodium selenite or 10 mg/kg of seleno-DL-methionine. Researchers in this study estimated that a 1000 g mallard consumes 100 g of feed per day. All 6 females and 5 of 6 males fed 100 mg/kg of sodium selenite died between days 16 and 39 of the study. None of these pairs reproduced during the study (Heinz *et al.* 1987). No effects on reproduction were found in mallards receiving 1, 5, or 10 mg/kg sodium selenite. Similarly, Heinz *et al.* (1989) found no significant difference in reproductive success between controls and mallards receiving 1, 2, or 4 mg/kg selenomethionine. Stanley *et al.* (1996) also found no difference in the reproductive success of mallards fed a diet containing 3.5 mg/kg seleno-DL-methionine.

Heinz *et al.* (1987) did not find significant difference in the fertility or proportion of eggs laid between their treatment groups of 1, 5, 10, and 25 mg/kg sodium selenite. However, hens in the 25 mg/kg group took longer to begin laying eggs and laid less frequently than the others. Laboratory studies using selenomethionine cite lower concentrations associated with adverse effects to reproduction. Mallards in the 10 mg/kg of seleno-DL-methionine group of the Heinz *et al.* (1987) study had lower hatching success (30.9%) compared to controls (65.7%) and the 10 mg/kg sodium selenite group (61.9%). Eight mg/kg selenomethione fed to female mallards accumulated to 3.5 mg/kg in the liver of hens and significantly reduced reproductive success (Heinz *et al.* 1989). Stanley *et al.* (1996) reported a decrease in reproductive success for mallards fed diets containing 7 mg/kg seleno-DL-methionine. Eggs produced at this concentration contained an average of 7.1 mg/kg selenium.

In a study on reproductive effects to black-crowned night herons, a diet containing 10 mg/kg dw seleno-DL-methionine did not significantly reduce hatching success

compared to controls. Researchers did note that femur lengths of dosed birds were shorter than controls. Eggs from herons dosed at 10 mg/kg dw seleno-DL-methionine contained an average 3.3 mg/kg selenium (Smith *et al.* 1988). A study using Easter screech-owls (*Otus asio*) had results similar to Smith *et al.* (1988). A diet containing 4.4 mg/kg ww (equivalent to 10 mg/kg dw) did not significantly reduce reproduction, although femur lengths of the young were shorter than controls (Wiemeyer and Hoffman 1996).

The number of abnormal embryos was not significantly different between controls and mallards at either 1 or 5 mg/kg of sodium selenite in the diet (Heinz *et al.* 1987). Groups receiving 10 or 25 mg/kg of sodium selenite or 10 mg/kg of seleno-DL-methionine produced 11.2, 22.2, and 18.3% abnormal embryos. The effects of sodium selenite were predominantly embryotoxic, such as stunted growth, swollen necks, and fewer than normal feathers. Seleno-DL-methionine related abnormalities were mostly teratogenic, for example, bill and eye defects, twisted legs and feet, and missing toes (Heinz *et al.* 1987). Egg concentrations of selenium in the group receiving 10 mg/kg of seleno-DL-methionine averaged 4.6 mg/kg (Heinz *et al.* 1987). Similar effects to reproduction have been observed in studies using dietary concentrations of 8, 15, and 16 mg/kg selenomethionine (Heinz *et al.* 1989; Heinz and Fitzgerald 1993).

Studies frequently note reduced survival of young exposed to selenium in the weeks that follow hatching. Heinz *et al.* (1987) found the number of 21-day old ducklings produced per hen was significantly lower for birds fed 25 mg/kg of sodium selenite or 10 mg/kg of seleno-DL-methionine. Hens fed 8 mg/kg selenomethionine in diet had an average of 4.6 ducklings survive to 6 days. This compared with 8.1, 8.5, 8.2, and 7.5 for hens with 0, 1, 2, or 4 mg/kg selenomethionine (Heinz *et al.* 1989).

The dietary threshold for effects to reproduction appears to lie between 4 and 8 mg/kg. Above this threshold, effects to reproduction include decreased hatching success, increased embryotoxicity, increased teratogenic effects, and decreased survival of young birds.

***Field Studies***

Laboratory studies often use doses that reflect levels available in the wild. As a result, field studies on the effects of selenium provide similar insights to its toxicity as do laboratory studies. Researchers have demonstrated a high risk of embryonic deformities in birds when population liver concentrations of selenium exceed 9 mg/kg (Heinz 1996). Wild populations with mean liver concentrations of selenium below 3 mg/kg are less likely to have a significant number of deformities.

Hoffman *et al.* (2002) collected American avocet (*Recurvirostra americana*) and black-necked stilt (*Himantopus mexicanus*) eggs from three separate field sites and hatched them in a laboratory. Hatching success and malformations did not differ between the sites. The highest egg concentrations were 31.4 mg/kg dw for avocets and 20.5 mg/kg dw for black-necked stilts. These concentrations did not significantly decrease hatching success or increase the number of malformations. Avocets did, however, have decreased embryo growth and lower long bone lengths at the highest concentration. These findings are comparable with those reported for black-crowned night herons and eastern screech owls (Smith *et al.* 1988; Wiemeyer and Hoffman 1996).

Survival and reproductive impacts to aquatic wild birds have been investigated by Ohlendorf *et al.* (1986a,b, 1988). Selenium concentrations in food ranged from 22 to 175 mg/kg dw. Clinical symptoms and effects to reproduction associated with

selenosis were observed in aquatic birds of the area and included adult emaciation, and embryonic and duckling malformations.

***Effects Metrics***

The most sensitive responses in birds exposed to selenium are associated with reproduction. None of the available reproduction studies included piscivorus bird species identified as focal species for this assessment or reasonable surrogates. Many of the studies used mallards. Their foraging behavior does not resemble the hypothetical piscivorus receptor. The hypothetical piscivorus receptor exhibits characteristics similar to brown pelican, osprey, belted kingfishers, and terns. It primarily forages on fish and lesser amounts of invertebrates. The lack of toxicity data for piscivorus birds exposed to selenium precludes the development of a dose-response curve (using either a single or multiple studies) or the derivation of a NOAEL and LOAEL for piscivorus birds (i.e., Options 1-3 in the hierarchy of decision criteria for choosing effects metrics are unavailable).

An insufficient amount of field data are available to develop field-based benchmarks for piscivorus birds of the Calcasieu Estuary, which eliminates Option 4 for choosing effects metrics.

The final option for choosing an effects metric for piscivorus birds exposed to selenium is to derive a range within which the threshold for this receptor group is expected to occur. The most sensitive reproductive response was observed in mallard ducks exposed to selenomethionine (Heinz *et al.* 1989). In this study, dietary concentrations above 4 mg selenomethione/kg resulted in significantly greater embryo malformations. Stanley *et al.* (1996) reported no reproductive effects from adult mallards dosed with 3.5 mg seleno-DL-methionine/kg. However, reduced hatching success and lower duckling weights occurred at 7 mg seleno-DL-methionine/kg.

Based on these studies, 4 mg selenomethionine/kg in diet was chosen as the lower bound of the threshold range for piscivorus birds exposed to selenium. This dietary concentration was multiplied by the food intake rate of mallard ducks (0.0554 kg/day, Nagy 1987) and normalized to the body weight female mallards for the 4 mg selenomethionine dose group (1.04 kg, Heinz *et al.* 1989) to derive the corresponding dose:

$$LT = \left( \frac{4.00\ mg\ Se}{kg\ diet} \times \frac{55.4\ g\ food}{day} \times \frac{1.00\ kg}{1000\ g} \right) / 1.04\ kg\ BW \qquad \textbf{EQUATION \#4}$$
$$= 0.214\ mg\ /\ kg\,bw\ /\ day$$

where *LT* is the lower threshold dose and *BW* is body weight.

Studies have shown black-crowned night herons and eastern screech owls to be less sensitive to selenium than mallards (Wiemeyer and Hoffman 1996; Smith *et al.* 1988). A dietary concentration of 10 mg selenomethionine/kg dw did not significantly reduce hatching success or increase the number of malformed embryos in these species. However, mallards fed this concentration exhibited adverse effects to reproduction (Heinz *et al.* 1987). Black-crowned night herons feed mainly on fish and invertebrates and therefore may be considered similar to the hypothetical piscivorus bird receptor. The hypothetical piscivorus bird likely has an upper threshold above mallard ducks, but within the range of the black-crowned night heron. It would seem reasonable then to select 10 mg selenomethionine/kg dw as the upper threshold of effects to piscivorus birds from selenium. This dietary concentration was multiplied by the food intake rate of black-crowned night herons (0.049 kg/day dw, calculated from Nagy 1987) and normalized to their body weight (0.883 kg, Dunning 1984) to derive the corresponding dose:

$$UT = \left( \frac{10.0\ mg\ Se}{kg\ diet} \times \frac{49.1\ g\ food}{day} \times \frac{1.00\ kg}{1000\ g} \right) / 0.883\ kg\ BW \quad \textbf{EQUATION \#5}$$
$$= 0.556\ mg / kg\,bw / day$$

where *UT* is the upper threshold dose and *BW* is body weight. Therefore, the threshold range for piscivorus birds exposed to selenium is 0.214 to 0.556 mg selenium/kg bw/day. The Appendix G benchmark for selenium is higher than the lower and upper toxicity thresholds that were selected for the piscivorus receptor. The benchmark was derived using higher food intake rates than those used in this assessment (Sample *et al.* 1996).

### 2.2.2.3 Mercury

Methylmercury is a strong nervous system toxicant. Its ability to cross the blood brain barrier results in brain lesions, damage to the central nervous system, and spinal cord degeneration (Wolfe *et al.* 1998). Methylmercury is absorbed into the bloodstream and transported to tissues and organs throughout the body (USEPA 1997b). As a result, neurological disorders, damage to organs, and effects on growth and development are characteristic effects of MeHg. Clinical symptoms of acute poisoning include ataxia, tremors, weakness in legs and wings, muscular incoordination, paralysis, recumbency, and convulsions (USEPA 1997b; Wolfe *et al.* 1998; Eisler 2000). Adverse effects also occur from chronic exposure to low concentrations of MeHg.

The reliance of piscivorus birds on fish makes them particularly susceptible to the adverse effects of MeHg toxicity. The proportion of mercury as MeHg in fish tissues

is generally greater than 90%; and increases with fish length, weight, and age (Eisler 2000). Concentration data are in wet weight (ww), unless noted otherwise.

***Survival***

Studies by Spalding *et al.* (2000a,b) and Bouton *et al.* (1999) found behavioral abnormalities and neurologic disturbances in great egrets (*Ardea albus*) dosed with 0.5 or 5 mg MeHgCl/kg. Birds had dingy feathers, avoided sun, and were less motivated to hunt. Great egrets in the high dose group experienced severe ataxia, as well as hematologic and histologic changes. Scheuhammer (1988) observed signs of mercury poisoning in Zebra finches (*Poephila guttata*) fed 5 mg MeHg/kg dry weight (dw). On day 40 of the 77-d study, some finches began exhibiting behavioral abnormalities. Symptoms included lethargy, fluffed feathers, and difficulty flying. The first death occurred on day 68 and by day 77 four of eight finches in the dose group had died with the rest showing neurological signs of mercury poisoning (Scheuhammer 1988). Neurological signs of poisoning did not appear until mercury concentrations were $15 mg/kg in the brain and between 30 and 40 mg/kg in the liver and kidneys. The high metabolism of the Zebra finch forces it to consume a greater amount of food, and as a result, mercury. This characteristic is similar to the belted kingfisher, which has a high food intake rate on a body weight basis (USEPA 1997b).

Neurological effects and death resulted from dietary treatments of 7.2 and 10 mg/kg of MeHg dicyandiamide fed to red-tailed hawks (*Buteo jamaicensis*; Fimreite and Karstad 1971). Birds that died showed symptoms similar to those described earlier: muscular weakness, in-coordination, weight loss. Liver mercury residues ranged from 17 to 20 mg/kg in chicks that died. Lesions of axons and myelin sheaths were found in all hawks fed 7.2 and 10 mg/kg (Fimreite and Karstad 1971). Hill and Soares (1984) found similar effects in coturnix (*Coturnix japonica*) at a diet of 8 mg MeHgCl/kg over a 9 wk period. During week 8, one male and three female birds

began losing muscular control. One female died during week 9 after displaying signs of severe mercury toxicity. No clinical signs were shown over the 9 wk study in coturnix fed diets containing 0.125 or 4 mg MeHgCl/kg (Hill and Soares 1984). Hill and Soares (1984) established a single oral dose $LD_{50}$ of 18 mg/kg body weight (bw) and an $LC_{50}$ of 47 mg/kg.

Borg *et al.* (1970) fed juvenile goshawks (*Accipiter g. gentilis l.*) liver and muscle from MeHg contaminated chickens. The MeHg concentration in chicken muscle was 10 mg/kg and in liver 40 mg/kg. Diets contained either contaminated muscle and liver for a concentration of 13 mg/kg or muscle only for an average concentration of 10 mg/kg. The estimated intake of MeHg by goshawks was 0.7-1.2 mg/kg/day. Symptoms of MeHg poisoning were observed after a 2 week latency period and included inappetence, muscular weakness, ataxia, and loss of body weight. Goshawks in the 13 mg/kg group died at 30, 38, and 47 d. One bird in the 10 mg/kg group died on day 39. MeHg concentrations in the brains of these birds ranged from 26 to 46 mg/kg and in the livers from 96 to 138 mg/kg (Borg *et al.* 1970). Concentrations similar to those used by Borg *et al.* (1970) also proved fatal to pheasants (Spann *et al.* 1972). In their study, pheasants fed 30 mg/kg ethyl mercury p-toluene, equivalent to 12.5 mg Hg/kg, died between 57 and 102 days of feeding (Spann *et al.*1972). Symptoms leading up to death were similar to previously mentioned studies.

***Reproduction***

Mercury's potent embryo toxicity makes reproduction one of the most sensitive endpoints. Mercury concentrations well below those required to cause effects in adults can negatively impact reproduction and survival of young (Scheuhammer 1988; USEPA 1997b). Adverse effects of mercury on reproduction include reduced hatchability caused by increased mortality of embryos, smaller clutch sizes, a greater

number of eggs laid outside the nest, and aberrant behavior (USEPA 1997b; Wolfe *et al.* 1998; Eisler 2000). Reproductive effects also extend to juvenile survival (Wolfe *et al.* 1998).

Adult mallard ducks (*Anas platyrhynchos*) were unaffected by mercury concentrations of 0.5 and 3 mg MeHg/kg dw, however, effects on reproduction were evident (Heinz 1979). Dosed hens laid more eggs outside the nestbox than controls, laid fewer sound eggs, and had less ducklings survive past one week (Heinz 1979). Duckling behavior was also affected. Treated ducklings had longer response times to tape recorded maternal calls than controls (Heinz 1979). Reproduction was similarly effected in black ducks (*Anas rubripes*) fed 3 mg MeHg dicyandiamide/kg over two reproductive seasons (Finley and Stendell 1978). The most harmful effects were on hatchability and duckling survival. Other effects were observed for clutch size, egg production, and the number of eggs incubated. Mercury concentrations in whole embryos that failed to hatch averaged 9.62 and 6.08 mg/kg for the first and second year, respectively. Brain mercury concentrations in dead ducklings ranged from 3.25 to 6.98 mg/kg and displayed lesions associated with mercury poisoning (Finley and Stendell 1978). Fimreite (1971) found comparable effects in pheasants fed 2-3 mg MeHg dicyandiamide/kg for 12 weeks. The number of shell-less eggs increased and egg weights decreased, as did hatchability and the number of fertilized eggs. Mercury concentrations in unhatched eggs ranged from 0.5 to 1.5 mg/kg (Fimreite 1971). A dietary dose of 10 mg/kg of ethyl mercury *p*-toluene (mercury equivalent of 4.2 mg/kg) reduced egg production 50-80% and increased mortality in eggs that were laid (Spann *et al.* 1972). A sample of treated eggs had an average mercury concentration of 1.5 mg/kg, and a range of 0.3 to 3.1 mg/kg (Spann *et al.* 1972).

### *Field Surveys*

Common loons (*Gavia immer*) in northwestern Ontario displayed reduced nest site fidelity and laid fewer eggs in areas where mercury concentrations in prey averaged >0.4 mg/kg. Adult loon brain concentrations of mercury between 2 and 3 mg/kg were also associated with adverse effects on reproductive behavior (Barr 1996). Monteiro and Furness (2001) observed no clinical signs of poisoning in a single oral dose experiment with MeHg on free-living Cory's Shearwater chicks (*Calonectris dimoedea*). Exposure levels ranged from 0.9 to 2.5 mg/kg body weight and the researchers noted that they were similar to the no adverse effects level (NOAEL) of 2.5 mg/kg found by Scheuhammer (1988). Monteiro and Furness (2001) estimated that the highest average mercury brain concentration in the experiment was 3.4 mg/kg. This concentration was based on a blood:brain ratio of 0.78 calculated from adult Cory's shearwaters concentrations.

### *Effects Metrics*

The most sensitive responses in birds exposed to methylmercury are associated with reproduction following long-term exposures. None of the available reproduction studies included species that could be considered piscivorus birds or reasonable surrogates. Mallards are often used in laboratory studies, however, their foraging behavior is considerably different from the hypothetical piscivorus receptor. The hypothetical piscivorus receptor bears much more of a resemblance to birds such as brown pelicans, osprey, belted kingfishers and terns. Its diet primarily consists of fish and lesser amounts of invertebrates. The lack of toxicity data for piscivorus birds exposed to methylmercury precludes the development of a dose-response curve (using either a single or multiple studies) or the derivation of a NOAEL and LOAEL for piscivorus birds (*i.e.*, Options 1-3 in the hierarchy of decision criteria for choosing effects metrics are unavailable).

No field data were available to develop field-based benchmarks for piscivorus birds, which eliminates Option 4 for choosing effects metrics.

The final option for choosing an effects metric for piscivorus birds exposed to methylmercury is to derive a range within which the threshold for this receptor group is expected to occur. The most sensitive reproductive response was observed in mallard ducks exposed to methylmercury for three generations (Heinz 1974, 1979; Heinz and Locke 1975). In this study, 0.5 mg Hg/kg (as methylmercury dicyanamide) led to small, but significant reductions in clutch size and duckling survival. Similarly, Fimreite (1971) estimated the threshold egg concentration for hatchability to be between 0.5 and 1.5 mg Hg/kg for ring-necked pheasants. It would therefore seem reasonable to select 0.5 mg Hg/kg in the diet as the lower bound of the threshold range for piscivorus birds exposed to methylmercury. This dietary concentration was multiplied by the food intake rate of mallard ducks (0.128 kg/day, as measured by Heinz 1979) and normalized to their body weight (1 kg, Heinz *et al.* 1989) to derive the corresponding dose:

$$LT = \left( \frac{0.500\ mg\ Hg}{kg\ diet} \times \frac{128\ g\ food}{day} \times \frac{1.00\ kg}{1000\ g} \right) / 1.00\ kg\ BW \qquad \textbf{EQUATION \#6}$$
$$= 0.0640\ mg / kg\,bw / day$$

where *LT* is the lower threshold dose and *BW* is body weight.

Survival and reproductive data on effects reveal a broad range of bird taxa that are severely affected by dietary concentrations of methylmercury ⁄ 10 mg/kg. None of the tested species, which included mallards (Heinz and Hoffman 1998), goshawks (Borg *et al.* 1970), ring-necked pheasants (Spann *et al.* 1972), white leghorn chickens (Scott 1977) and Japanese quail (Hill and Soares 1984; Scott 1977), were able to tolerate dietary concentrations of methylmercury close to or greater than 10 mg/kg.

The highest level of methylmercury in the diet that did not cause adverse impacts to a test species was 6 mg/kg. The test species was red-tailed hawks (Fimreite and Karstad 1971). It would therefore seem reasonable to select 6 mg Hg/kg in the diet as representing the tolerant end of the threshold range for piscivorus birds exposed to methylmercury. This dietary concentration was multiplied by the food intake rate of red-tailed hawks (0.109 kg/day; Craighead and Craighead 1969) and normalized to their body weight (1.126 kg; Dunning 1984) to derive the corresponding dose:

$$UT = \left( \frac{6.00\ mg\ Hg}{kg\ diet} \times \frac{109\ g\ food}{day} \times \frac{1.00\ kg}{1000\ g} \right) / 1.13\ kg\ BW \qquad \textbf{EQUATION \#7}$$
$$= 0.581\ mg\ /\ kg\,bw\ /\ day$$

where *UT* is the upper threshold dose and *BW* is body weight. Therefore, the threshold range for piscivorus birds exposed to methylmercury is 0.0640 to 0.581 mg Hg/kg bw/day.

***Total PCBs***

Commercial PCB mixtures elicit a variety of toxic effects, but some specific coplanar PCB congeners cause toxic responses through the induction of mixed function oxidase enzymes, such as aryl hydrocarbon hydroxylase (AHH) and 7-ethoxyresorufin-O-deethylase (EROD; Powell *et al.* 1997; Metcalfe and Haffner 1995). The effects range from decreased body weight to reproductive impairment to mortality (CCME 1999). Piscivorus birds are exposed to PCBs primarily through the consumption of prey that has accumulated these substances. Birds have been shown to efficiently absorb PCBs from feed (Drouillard and Norstrom 2000; Drouillard and Norstrom 2001; Drouillard *et al.* 2001) and PCBs are transferred from adult females to eggs at predictable rates (Bargar *et al.* 2001). Froese *et al.* (1998) and Secord *et al.* (1999) studied the accumulation of PCBs in tree swallow eggs and nestlings from prey. Custer *et al.* (1997; 1999) and Larson *et al.* (1996) reported substance

concentrations in cormorant eggs and embryos. These studies and others (e.g., Environment Canada 1998; Hoffman *et al.* 1996b; Eisler and Belisle 1996) have shown that PCBs accumulate in a variety of avian species to concentrations that may cause adverse effects.

***Mortality***

Heath *et al.* (1972) studied a number of avian species and their sensitivities to oral doses of PCBs. Bobwhite quail, Japanese quail, mallard duck, and ring-necked pheasants were all treated with oral doses of Aroclor 1254 for 5 days and observed for mortality. The most sensitive of the species was the bobwhite quail, with a median lethality response to a dietary PCB concentration of 604 mg/kg. The other species tested were less sensitive, with oral $LC_{50}$s of 2898, 2699, and 1091 mg/kg diet respectively. Pheasants fed Aroclor 1254 by capsule at a rate of 210 mg/day died within 3.8 days on average, while those fed 20 mg/day died within an average of 46 days (Dahlgren et al 1972a). The brains of these pheasants were analyzed immediately after death and showed total PCB concentrations ranging from 320 to 770 mg/kg ww. The investigators also sacrificed some of the birds just prior to death and compared the brain tissue PCB residues of these samples with those of the birds sampled after death. The authors concluded that PCB concentrations of 300-400 mg/kg ww in brain tissue correlated well with mortality. Bird *et al.* (1978) noted a significant increase in chick mortality at an Aroclor 1254 dietary concentration of 20 mg/kg and adipose tissue concentrations of 20 mg/kg ww. Platonow *et al.* (1973) found dietary treatments of 500 mg/kg of Aroclor 1254 fed to day old cockerels caused mortality of half of the birds within 43 days. Their mortality, as well as edema, hemorrhaging, and liver and kidney necrosis, was associated with brain tissue concentrations of 120 mg/kg ww. Prestt *et al.* (1970) found a median lethal dietary dose rate of Aroclor 1254 to adult Bengalese finches to be 256 mg/kg/day. These birds acquired brain tissue concentrations of 290 mg/kg ww and experienced enlarged

kidneys and hydropericardium. Stickel *et al.* (1984) reviewed the existing literature regarding PCB tissue concentrations and associated morality rates, and expanded the set to include grackles, red-winged blackbirds, brown headed cow birds, and starlings. The results of this effort indicated that brain tissue PCB residues greater than 310 mg/kg ww were associated with an increased likelihood of death due to PCB poisoning.

Heath *et al.* (1972) investigated the toxicities of various Aroclor mixtures to bobwhite quail, Japanese quail, mallard duck, and ring-necked pheasants. Aroclor 1248 was consistently the least toxic of three Aroclor mixtures (1248, 1254, and 1260) tested, with oral $LC_{50}$s of 1175, 4844, 2798, and 1312 mg/kg diet, for bobwhite quail, Japanese quail, mallard duck, and ring-necked pheasant, respectively. Bobwhite quail and ring-necked pheasants were the most sensitive to the 1254 ($LC_{50}$s of 604 and 1091 mg/kg diet) and 1260 ($LC_{50}$s of 747 and 1260 mg/kg diet) Aroclor mixtures.

***Reproduction***

Reproductive effects in birds caused by PCB exposure include reduced egg productivity, egg hatchability, and chick growth rates (CCME 1999). Dietary and egg injection studies are most common, as well as field studies examining egg and hatchling concentrations and hatching success. Of the species studied, chickens appear to be the most sensitive, followed by pheasants and turkey, ducks and then herring gulls (Bosveld and van den Berg 1994). PCBs appear to have no adverse effects on total egg weight, eggshell weight, or eggshell thickness (Lillie *et al.* 1974; Britton and Huston 1973; Scott 1977).

Dietary exposures of chickens to PCB formulations 1242, 1248, and 1254 at 20 mg/kg feed for 9 weeks resulted in decreased egg production (Lillie *et al.* 1974). Average egg production over the course of the trials decreased from 79% hen day-egg

production in controls to 67.5% in the Aroclor 1242 and 1248 treatments and 71.3% in the Aroclor 1254 treatment. Scott (1977) provided hens a diet with 20 mg/kg PCB 1248 in feed and reported reduced hen day-egg production to 64.8% compared to 74.5% for controls, results similar to Lillie *et al.* (1974).

The effects of Aroclor 1254 on the hatchability of eggs from white leghorn hens was investigated by Platonow and Reinhart (1973) and Lillie *et al.* (1974). In both studies, groups of birds were orally exposed to PCBs in their feed in two treatments – one study using 2 and 20 mg/kg diet for 9 weeks and the other 5 and 50 mg/kg diet for 14 weeks. Results after the treatment periods showed significant reduction in both hatchability and egg production at the highest concentrations in each study. The 20 mg/kg treatment reduced hatchability to 69% and the 50 mg/kg treatment reduced hatchability to near 0%. Tumasonis *et al.* (1973) also reported significant reduction in the hatchability of white leghorn hens' eggs (to 34%) after 2 weeks of exposure to drinking water treated with 50 mg/L Aroclor 1254, and 0% hatchability after 3 weeks. Platonow and Reinhart (1973) tested the same concentration, but in food (50 mg/kg), and saw a similarly sudden response, but Lillie *et al.* (1974), treating with a lower concentration, reported a more delayed effect. Pheasants fed 12.5 mg Aroclor 1254 weekly by gavage (Dahlgren *et al.* 1972b) showed a significantly decreased hatching rate of 50% compared to controls in one experiment, but insignificant changes in another. Ring doves fed a diet containing 10 mg/kg Aroclor 1254 experienced near total reproductive failure six months later with only 2 of 20 eggs hatching and fledging and one pair laying no eggs (Peakall *et al.* 1972). Cytogenic studies on the embryos revealed increased chromosome aberration rates, suggesting that PCBs have a clastogenic, or chromosome breaking, action.

Scott (1977) and Lillie *et al.* (1974) studied the effects Aroclor 1248 on the hatchability of hen eggs. Eight week dietary treatments of 10 mg/kg feed reduced egg

hatchability 50% (Scott 1977). The response was even more pronounced at higher dietary levels, with 20 mg/kg feed reducing egg hatchability to 2.4 % compared to controls. Lillie *et al.* (1974) provided diets containing 20 mg/kg Aroclor 1248 as well and observed that hen egg hatchability was reduced to 1.8% for the 9$^{th}$ week of the exposure, while average egg hatchability for the 9 week period was 11.9%. The hatching rate for the control group was in this study was 93.7%.

Britton and Huston (1973) observed the effects of Aroclor 1242 on hen egg hatchability and found the results similar to those of Aroclor 1254. The percent egg hatchability decreased to 75% following a 6 week maternal dietary concentration of 10 mg/kg in feed and diets containing 20 mg/kg and 40 mg/kg reduced hatchability to 50% and 0% respectively. Assuming a feed consumption rate of 6% of body weight daily (CCME 1993; Lillie *et al.* 1974), this translates to daily doses of 0.6, 1.2 and 2.4 mg/kg bw/d. Egg yolk concentrations of Aroclor 1242 were also monitored in the investigation. The authors reported that the statistical significance of the reduced hatchability coincided with egg yolk concentrations ranging from 2.4 to 5.6 mg/kg ww.

Fernie *et al*. (2001a; 2001b) investigated the effects of PCBs to American kestrels using a combination of Aroclor mixtures (1:1:1, Aroclor 1248:1254:1260) in the diet. First generation kestrels were exposed to the PCB mixture in the diet, with 0.100 mL aliquots of 4.85 mg/g PCBs in safflower oil injected into the brain of dead day-old cockerels. Kestrels had been observed to preferentially eat the head of cockerels, and, assuming they consumed all of the substance, and that kestrels ate 1.5 cockerels per day, an approximate daily intake rate of 7 mg/kg bw/d was reportedly achieved for this study. Whole egg concentrations of 34 mg/kg ww were reached in the treatment group, compared to 0 mg/kg in the control group. The second generation kestrels, receiving the PCB exposure *in ovo*, were raised to adults and mated with unexposed

birds and their mating success monitored. These birds experienced a laying lag, had smaller clutches and produced fewer fertile eggs, hatchlings and fledglings per breeding pair. Pairs with a PCB exposed female experienced complete mortality in 25% of broods, while pairs with a PCB exposed male experienced complete mortality in 63% of broods.

***Other effects***

PCBs have been reported to induce a number of sublethal effects, including increased EROD and AHH activity, liver necrosis, behavioral changes, decreased weight gain, changes in organ weights, edema, and growth of bird chicks (Hoffman *et al.* 1996b; Eisler and Belisle 1996; Environment Canada 1998). One day old New Hampshire X white leghorn chickens were fed 3.2 mg/kg bw/day Aroclor 1248 in their diet for 35 days (Rehfeld *et al.* 1972a) and significantly reduced growth rates observed. Liver enlargement and spleen atrophy were also reported. Rehfeld *et al.* (1972b) also examined the growth of chicks exposed over 42 days and found that daily doses of 2.5 mg/kg bw significantly reduced body weight gain. Chicks were also noted to experience reduced body weight gains from PCBs in the maternal diet (Lillie *et al.* 1974). Feed concentrations of 20 mg/kg (1.2 mg/kg bw/d) Aroclors 1232 and 1242 and 2 mg/kg (0.12 mg/kg bw/d) Aroclors 1254 and 1260 significantly reduced three-week body weight gains in newly hatched chicks. Heinz *et al.* (1980) reported significant weight loss (-7%) in adult ring doves at an Aroclor 1254 dose level of 7.2 mg/kg bw/d. Drouillard *et al.* (2001b) reported no significant effects to kestrel body weights after dosing with an Aroclor mixture (1248:1254:1260 at a ratio of 1:1:1) for 100 days at an estimated dose level of 7 mg/kg bw/d.

Yamashita *et al.* (1993) reported an increase in embryonic deformity rates in double-crested cormorants in areas of the Great Lakes contaminated with PCBs. Birds have experienced three- and five-fold increases in the number of deformed embryos in

association with total PCB concentrations in eggs of 6.8 and 11 mg/kg respectively. This is in comparison to a reference site with egg concentrations of 3.6 mg/kg egg.

PCB exposure has also been associated with altered behavior among birds. First generation ring doves were maintained on a diet containing 10 mg/kg Aroclor 1254 and their progeny were raised and mated (Peakall and Peakall 1973). The attentiveness of the second generation birds to their eggs, as measured by egg temperature, was affected adversely. Temperature readings of the PCB treated group showed a lower mean egg temperature and greater temperature variation, indicating that the parents spent less time incubating. This erratic behavior may explain the reduced hatching and fledging success of the third generation birds from this investigation. McCarty and Secord (1999a) have shown that nest building behavior may also be adversely affected by PCB exposure. Tree swallows inhabiting sites along the PCB-contaminated Hudson River, N.Y. produced nests of poorer quality compared to reference sites, as measured by nest mass and the number of feathers lining the nest. Nests along the Hudson River had significantly lower weights and significantly fewer feathers lining the nests. As female swallows build most of the nest cup, and males gather most of the feathers for the lining, both sexes appear to be affected by PCB exposure. High frequency of nest abandonment and burying eggs beneath nest material was also observed in Hudson River tree swallow nests (McCarty and Secord 1999b) compared to reference sites.

***Field studies***

Bosveld and van den Berg (1994) conducted a ten year review of total PCBs in wild bird eggs and found that bird eggs from contaminated sites generally contained less than 20 mg/kg ww total PCBs, while reference sites ranged from 2.0 to 3.0 mg/kg. One exception to this was the herring gull, whose eggs had much higher levels of PCBs, up to 100 mg/kg ww.

Tree swallows feed on emerging insects (Blancher and McNicol 1991) and thus PCB residues in tree swallow eggs and nestling tissues may represent trophic transfer of these substances from sediments (Fairchild *et al.* 1992; Bishop *et al.* 1999). PCB concentrations in tree swallows taken from the Hudson River area in New York were among the highest ever reported in that species (McCarty and Secord 1999b). These concentrations reached as high as 100 mg/kg ww egg with mean concentrations ranging from 6 to 30 mg/kg ww egg. At this site, hatchability of eggs and total reproduction (fledglings produced per egg laid) were significantly reduced in the study areas in 1994 compared to a 1991 reference site. Measurements taken in 1995, however, showed no significant differences in hatchability or reproduction between study and reference sites, despite similar total PCB concentrations.

Other field studies have also shown no significant differences in reproductive success between contaminated and reference sites. Bishop *et al.* (1999) studied tree swallow reproduction along the Great Lakes and St. Lawrence River with a reference site in Georgian Bay. Total PCB concentrations in the study sites were 2 to 13 times higher than in the reference site. EROD activity, vitamin A levels and porphyrin concentrations were all affected in birds in the study sites, relative to reference sites, but hatching and fledging success were unaffected. Custer *et al.* (1998) observed tree swallows in the Fox River and lower Green Bay region in Wisconsin. Mean total PCB concentrations in pippers and nestlings taken from four study sites ranged from 0.05 to 3.85 mg/kg ww. EROD activity showed a positive correlation with PCB concentration, but hatching success at the sites was unrelated to PCB concentrations in pippers or food.

Soil samples from the Crab Orchard National Wildlife Refuge in Illinois contained Aroclor 1254 at levels as high as 120,000 mg/kg (Arena *et al.* 1999). Investigators found significantly decreased fledging success and nest attentiveness in starlings at

the study sites compared to reference sites with no known PCB contamination. A relationship was also observed between the concentrations of Aroclor 1254 in nestling carcasses and animal matter taken from their stomachs. Hoffman *et al.* (1993) reported reduced hatching success in common terns in Saginaw Bay, WI. where eggs accumulated total PCBs of 8.5 mg/kg egg ww compared to a reference site with total PCB levels of 4.7 mg/kg egg ww. These eggs, however, were also known to contain DDE and mercury, although concentrations of these substances did not vary between the study and reference sites.

Cormorant egg hatchability in the Netherlands was negatively correlated to PCBs in eggs (Dirksen *et al.* 1995) where the mean concentration of the sum of 6 PCB congeners (28, 52, 101, 138, 153, and 180) was 21 mg/kg ww in cormorant eggs at the most contaminated site and hatchability was reduced to 0.7 hatched eggs per clutch. However, these effects also correlated with DDE concentrations in cormorant eggs. Egg hatchability and fledgling success of cormorants was significantly lower in a colony with higher PCB concentrations in yolk sacs (van den Berg *et al.* 1994). Individual PCB congeners were measured in this study and mean concentrations of 11 common congeners in the yolk sacs of unhatched cormorant embryos ranged from 3 to 350 mg/kg lipid weight.

***Effects Metrics***

The most sensitive responses in birds exposed to total PCBs are associated with reproduction following long-term exposures. None of the available laboratory reproduction studies included species that could be considered piscivorus birds or reasonable surrogates. The lack of toxicity data for piscivorus birds exposed to total PCBs precludes the development of a dose-response curve (using either a single or multiple studies) or the derivation of a NOAEL and LOAEL for piscivorus birds (i.e.,

options 1-3 in the hierarchy of decision criteria for choosing effects metrics are unavailable).

No field data were available to develop field-based benchmarks for piscivorus birds, which eliminates Option 4 for choosing effects metrics.

The final option for choosing an effects metric for piscivorus birds exposed to PCBs is to derive a range within which the threshold for this receptor group is expected to occur. The most sensitive reproductive response was observed in white leghorn chickens (Lillie *et al.* 1974) exposed to Aroclor 1254 for 63 days. Hens were given commercial feed with 2 mg/kg PCBs. Assuming hens consume 6% of their body weight in feed per day (CCME 1993; Lillie *et al.* 1974), the hens received a PCB dose of approximately 0.12 mg/kg bw/d. This exposure resulted in no significant effects on fertility, egg production, shell thickness, or hatchability, but did significantly reduce the growth rate of chicks. The three week weight gain for the control group was 163 g while the treatment group gained 151 g. Similarly, Platonow and Reinhart (1973) exposed white leghorn chickens to Aroclor 1254 via feed for 98 days. Using the same 6% consumption rate, birds given dietary concentrations of 5 mg/kg PCBs received daily doses of approximately 0.3 mg/kg bw/d. This treatment level caused no adverse effects to hen day egg production, egg fertility, or egg hatchability. When the study was extended, however, to 273 days, both egg production and fertility decreased significantly.

Ring-necked pheasants have been shown to be somewhat less sensitive to PCBs than chickens, as reported by Dahlgren *et al.* (1972b). Pheasant hens were given weekly doses of Aroclor 1254 by glass tube into the esophagus for 17 weeks. A weekly treatment of 12.5 mg/bird/week, combined with an assumed body weight of 1 kg per bird (USEPA 1995), translates to an estimated daily dose of 1.8 mg/kg bw/d. This

treatment level caused no impairment of reproductive success in terms of egg production, while observations of egg fertility, hatchability, and chick survival to 6 weeks was inconclusive.

Fernie *et al.* (2001b) dosed kestrels with a mixture of Aroclors (1248:1254:1260 1:1:1) for 100 days to examine reproductive effects of PCBs. To achieve this dose, 0.100 mL aliquots of 4.85 mg/g PCBs in safflower oil were injected in the brain of dead day-old cockerels and then fed to the kestrels. Kestrels had been observed to preferentially eat the head of cockerels, and, assuming they ate the entire head, and that the kestrels ate 1.5 cockerels per day, an approximate daily intake rate of 7 mg/kg bw/d was reportedly achieved for this study. The birds in this study experienced a significant increase in laying lag (20.8 days compared to 14.5 days for controls) and other reproductive measures of effect, including clutch size ($p=0.08$) and number of fertile eggs ($p=0.14$), hatchlings ($p=0.06$), and fledglings ($p=0.05$) per breeding pair, decreased as well, but not significantly. The second generation kestrels, receiving the PCB exposure *in ovo*, were raised to adults and mated with unexposed birds and their mating success monitored. These birds experienced a laying lag, had smaller clutches and produced fewer fertile eggs, hatchlings and fledglings per breeding pair. Pairs with PCB-exposed females experienced complete mortality in 25% of broods within 6 days of hatching, while pairs with PCB-exposed males experienced complete mortality in 63% of broods. The number of fertile eggs and the number of hatchlings per breeding pair, however, were slightly higher in treatment groups than in controls. Because the effects were fairly minor and not expressed until the second generation, we selected 7 mg PCB/kg bw/d to be the tolerant end of the threshold range for piscivorus birds exposed to PCBs.

Therefore, the threshold range for piscivorus birds exposed to PCBs is 0.12 to 7.0 mg total PCBs/kg bw/day.

### 2.2.3 Risk Characterization

In the risk characterization phase of the probabilistic risk assessment, the results of the exposure assessment (i.e., reverse cumulative distribution functions) and effects measures were integrated to develop risk estimates for each COC in each AOC. Ideally, risk characterization involves three major lines of evidence: comparison of modeled exposure to lab-derived effects metrics, *in situ* or whole-media toxicity tests, and biological surveys. For piscivorus birds, however, the latter two lines of evidence are not available. We therefore rely on the risk estimates generated from the comparison of modeled exposure to effects thresholds derived from laboratory studies.

## 3.0 Results and Discussion

## 3.1 Probabilistic Ecological Risk Assessment

***TCDD-TEQs – Bayou d'Inde AOC***

The Monte Carlo analysis revealed that exposure of average-sized piscivorus birds to TCDD-TEQs in BI AOC could range from a minimum of 3.70 to a maximum of 82.6 ng/kg bw/day. The mean exposure is 18.8 ng/kg bw/day and the median exposure is 17.1 ng/kg bw/day. Ninety percent of exposure estimates are between of 8.75 and 34.8 ng/kg bw/day. Figure H3-4 depicts the cumulative distribution of TCDD-TEQs intake rates for the hypothetical average-sized piscivorus bird species.

Sensitivity analysis revealed that the FMR power term was the most important variable [Pearson correlation coefficient ($r_p$) = 0.61] followed by FMR slope ($r_p$ = 0.53) and gross energy of fish ($r_p$ = -0.44).

The probability bounds estimated for average-sized piscivorus birds are depicted in Figure H3-4. The 10$^{th}$ percentile of the probability envelope formed by the lower and upper bounds ranges between 5.31 and 28.5 ng/kg bw/day. The 50$^{th}$ percentile ranges between 10.47 and 48.4 ng/kg bw/day, and the 90$^{th}$ percentile ranges between 17.4 and 103 ng/kg bw/day. In comparison, the 10$^{th}$ percentile of the Monte Carlo prediction is 10.0, the 50$^{th}$ percentile is 16.9, and the 90$^{th}$ percentile is 29.5 ng/kg bw/day.

The Monte Carlo analysis revealed that exposure of small piscivorus bird species to TCDD-TEQs could range from a minimum of 11.6 to a maximum of 191 ng/kg bw/day. The mean exposure is 42.6 ng/kg bw/day and the median exposure is 39.6 ng/kg bw/day. Ninety percent of exposure estimates are between 22.6 and 72.8 ng/kg bw/day. Figure H3-5 shows the cumulative distribution of TCDD-TEQs intake rates for small piscivorus bird species in BI AOC.

Sensitivity analysis revealed that the FMR slope was the most important variable [Pearson correlation coefficient ($r_p$) = 0.63], followed by gross energy of fish ($r_p$ = -0.53) and FMR power term ($r_p$ = 0.38).

The probability bounds estimated for small piscivorus birds are depicted in Figure H3-5. The 10$^{th}$ percentile of the probability envelope formed by the lower and upper bounds ranges between 13.7 and 69.7 ng/kg bw/day. The 50$^{th}$ percentile ranges between 24.7 and 111 ng/kg bw/day, and the 90$^{th}$ percentile ranges between 38.3 and

217 ng/kg bw/day. In comparison, the 10th percentile of the Monte Carlo prediction is 25.6, the 50th percentile is 39.5, and the 90th percentile is 63.2 ng/kg bw/day.

***TCDD-TEQs – Middle Calcasieu River AOC***

The Monte Carlo analysis revealed that exposure of average-sized piscivorus birds to TCDD-TEQs in the MCR AOC could range from a minimum of 1.59 to a maximum of 34.7 ng/kg bw/day. The mean exposure is 7.27 ng/kg bw/day and the median exposure is 6.56 ng/kg bw/day. Ninety percent of exposure estimates are between of 3.31 and 13.6 ng/kg bw/day. Figure H3-6 depicts the cumulative distribution of TCDD-TEQs intake rates for the hypothetical average-sized piscivorus bird species in the MCR AOC.

Sensitivity analysis revealed that the FMR power term was the most important variable [Pearson correlation coefficient ($r_p$) = 0.61] followed by FMR slope ($r_p$ = 0.53) and gross energy of fish ($r_p$ = -0.45).

The probability bounds estimated for average-sized piscivorus birds are depicted in Figure H3-6. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 1.14 and 19.2 ng/kg bw/day. The 50th percentile ranges between 3.62 and 38.7 ng/kg bw/day, and the 90th percentile ranges between 7.33 and 134 ng/kg bw/day. In comparison, the 10th percentile of the Monte Carlo prediction is 3.81, the 50th percentile is 6.54, and the 90th percentile is 11.5 ng/kg bw/day.

The Monte Carlo analysis revealed that exposure of small piscivorus bird species to TCDD-TEQs could range from a minimum of 4.77 to a maximum of 82.1 ng/kg bw/day. The mean exposure is 16.5 ng/kg bw/day and the median exposure is 15.3 ng/kg bw/day. Ninety percent of exposure estimates are between 8.55 and 28.5 ng/kg

bw/day. Figure H3-7 shows the cumulative distribution of TCDD-TEQs intake rates for small piscivorus bird species in the MCR AOC.

Sensitivity analysis revealed that the FMR slope was the most important variable [Pearson correlation coefficient ($r_p$) = 0.63], followed by gross energy of fish ($r_p$ = --0.53) and FMR power term ($r_p$ = 0.38).

The probability bounds estimated for small piscivorus birds are depicted in Figure H3-7. The 10$^{th}$ percentile of the probability envelope formed by the lower and upper bounds ranges between 3.02 and 42.0 ng/kg bw/day. The 50$^{th}$ percentile ranges between 9.02 and 84.8 ng/kg bw/day, and the 90$^{th}$ percentile ranges between 18.5 and 277 ng/kg bw/day. In comparison, the 10$^{th}$ percentile of the Monte Carlo prediction is 9.64, the 50$^{th}$ percentile is 15.2, and the 90$^{th}$ percentile is 24.7 ng/kg bw/day.

***TCDD-TEQs – Reference Areas***

The Monte Carlo analysis revealed that exposure of average-sized piscivorus birds to TCDD-TEQs in the reference areas could range from a minimum of 1.13 to a maximum of 27.1 ng/kg bw/day. The mean exposure is 5.74 ng/kg bw/day and the median exposure is 5.20 ng/kg bw/day. Ninety percent of exposure estimates are between of 2.67 and 10.7 ng/kg bw/day. Figure H3-8 depicts the cumulative distribution of TCDD-TEQs intake rates for the hypothetical average-sized piscivorus bird species.

Sensitivity analysis revealed that the FMR power term was the most important variable [Pearson correlation coefficient ($r_p$) = 0.61] followed by FMR slope ($r_p$ = 0.53) and gross energy of fish ($r_p$ = -0.44).

The probability bounds estimated for average-sized piscivorus birds are depicted in Figure H3-8. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 1.08 and 18.4 ng/kg bw/day. The 50th percentile ranges between 3.47 and 37.6 ng/kg bw/day, and the 90th percentile ranges between 7.15 and 132 ng/kg bw/day. In comparison, the 10th percentile of the Monte Carlo prediction is 3.05, the 50th percentile is 5.20, and the 90th percentile is 9.01 ng/kg bw/day.

The Monte Carlo analysis revealed that exposure of small piscivorus bird species to TCDD-TEQs could range from a minimum of 3.55 to a maximum of 58.6 ng/kg bw/day. The mean exposure is 13.1 ng/kg bw/day and the median exposure is 12.1 ng/kg bw/day. Ninety percent of exposure estimates are between 6.94 and 22.3 ng/kg bw/day. Figure H3-9 shows the cumulative distribution of TCDD-TEQs intake rates for small piscivorus bird species in the reference areas.

Sensitivity analysis revealed that the FMR slope was the most important variable [Pearson correlation coefficient ($r_p$) = 0.63], followed by gross energy of fish ($r_p$ = -0.53) and FMR power term ($r_p$ = 0.38).

The probability bounds estimated for small piscivorus birds are depicted in Figure H3-9. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 2.85 and 40.1 ng/kg bw/day. The 50th percentile ranges between 8.67 and 82.4 ng/kg bw/day, and the 90th percentile ranges between 18.1 and 274 ng/kg bw/day. In comparison, the 10th percentile of the Monte Carlo prediction is 7.83, the 50th percentile is 12.0, and the 90th percentile is 19.3 ng/kg bw/day.

***Selenium – Bayou d'Inde AOC***

The Monte Carlo analysis revealed that exposure of average-sized piscivorus birds to selenium in BI AOC could range from a minimum of 0.0246 to a maximum of 0.554

mg/kg bw/day. The mean exposure is 0.125 mg/kg bw/day and the median exposure is 0.113 mg/kg bw/day. Ninety percent of exposure estimates are between of 0.0583 and 0.232 mg/kg bw/day. Figure H3-10 depicts the cumulative distribution of selenium intake rates for the average-sized piscivorus bird species.

Sensitivity analysis revealed that the FMR power term was the most important variable [Pearson correlation coefficient ($r_p$) = 0.61] followed by the FMR slope ($r_p$ = 0.53) and gross energy of fish ($r_p$ = -0.44).

The probability bounds estimated for average-sized piscivorus birds are depicted in Figure H3-10. The 10$^{th}$ percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0435 and 0.144 mg/kg bw/day. The 50$^{th}$ percentile ranges between 0.0795 and 0.254 mg/kg bw/day, and the 90$^{th}$ percentile ranges between 0.138 and 0.496 mg/kg bw/day. In comparison, the 10$^{th}$ percentile of the Monte Carlo prediction is 0.0670, the 50$^{th}$ percentile is 0.113, and the 90$^{th}$ percentile is 0.197 mg/kg bw/day.

The Monte Carlo analysis revealed that exposure of small piscivorus birds to selenium in BI AOC could range from a minimum of 0.0778 to a maximum of 1.25 mg/kg bw/day. The mean exposure is 0.284 mg/kg bw/day and the median exposure is 0.264 mg/kg bw/day. Ninety percent of exposure estimates are between of 0.151 and 0.486 mg/kg bw/day. Figure H3-11 depicts the cumulative distribution of selenium intake rates for the small piscivorus bird species.

Sensitivity analysis revealed that the FMR slope was the most important variable [Pearson correlation coefficient ($r_p$) = 0.63] followed by the gross energy of fish ($r_p$ = -0.53) and FMR power term ($r_p$ = 0.38).

The probability bounds estimated for small piscivorus birds are depicted in Figure H3-11. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.111 and 0.356 mg/kg bw/day. The 50th percentile ranges between 0.186 and 0.587 mg/kg bw/day, and the 90th percentile ranges between 0.302 and 1.06 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo prediction is 0.170, the 50th percentile is 0.263, and the 90th percentile is 0.420 mg/kg bw/day.

***Selenium – Reference Areas***

The Monte Carlo analysis revealed that exposure of average-sized piscivorus birds to selenium in the reference areas could range from a minimum of 0.0247 to a maximum of 0.543 mg/kg bw/day. The mean exposure is 0.120 mg/kg bw/day and the median exposure is 0.109 mg/kg bw/day. Ninety percent of exposure estimates are between of 0.0559 and 0.222 mg/kg bw/day. Figure H3-12 depicts the cumulative distribution of selenium intake rates for the average-sized piscivorus bird species.

Sensitivity analysis revealed that the FMR power term was the most important variable [Pearson correlation coefficient ($r_p$) = 0.61] followed by the FMR slope ($r_p$ = 0.53) and gross energy of fish ($r_p$ = -0.44).

The probability bounds estimated for average-sized piscivorus birds are depicted in Figure H3-12. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0342 and 0.170 mg/kg bw/day. The 50th percentile ranges between 0.0664 and 0.290 mg/kg bw/day, and the 90th percentile ranges between 0.111 and 0.608 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo prediction is 0.0639, the 50th percentile is 0.108, and the 90th percentile is 0.190 mg/kg bw/day.

The Monte Carlo analysis revealed that exposure of small piscivorus birds to selenium in the reference areas could range from a minimum of 0.0756 to a maximum of 1.26 mg/kg bw/day. The mean exposure is 0.273 mg/kg bw/day and the median exposure is 0.254 mg/kg bw/day. Ninety percent of exposure estimates are between of 0.146 and 0.466 mg/kg bw/day. Figure H3-13 depicts the cumulative distribution of selenium intake rates for the small piscivorus bird species.

Sensitivity analysis revealed that the FMR slope was the most important variable [Pearson correlation coefficient ($r_p$) = 0.63] followed by the gross energy of fish ($r_p$ = -0.53) and FMR power term ($r_p$ = 0.38).

The probability bounds estimated for small piscivorus birds are depicted in Figure H3-13. The 10$^{th}$ percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0880 and 0.417 mg/kg bw/day. The 50$^{th}$ percentile ranges between 0.157 and 0.666 mg/kg bw/day, and the 90$^{th}$ percentile ranges between 0.245 and 1.29 mg/kg bw/day. In comparison, the 10$^{th}$ percentile of the Monte Carlo prediction is 0.162, the 50$^{th}$ percentile is 0.252, and the 90$^{th}$ percentile is 0.404 mg/kg bw/day.

***Mercury – Bayou d'Inde AOC***

The Monte Carlo analysis revealed that exposure of average-sized piscivorus birds to mercury in BI AOC could range from a minimum of 0.00785 to a maximum of 0.177 mg/kg bw/day. The mean exposure is 0.0394 mg/kg bw/day and the median exposure is 0.0356 mg/kg bw/day. Ninety percent of exposure estimates are between of 0.0183 and 0.0731 mg/kg bw/day. Figure H3-14 depicts the cumulative distribution of mercury intake rates for the average-sized piscivorus bird species.

Sensitivity analysis revealed that the FMR power term was the most important variable [Pearson correlation coefficient ($r_p$) = 0.61] followed by FMR slope ($r_p$ = 0.53) and gross energy of fish ($r_p$ = -0.44).

The probability bounds estimated for average-sized piscivorus birds are depicted in Figure H3-14. The 10$^{th}$ percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0124 and 0.0473 mg/kg bw/day. The 50$^{th}$ percentile ranges between 0.0231 and 0.0820 mg/kg bw/day, and the 90$^{th}$ percentile ranges between 0.0395 and 0.163 mg/kg bw/day. In comparison, the 10$^{th}$ percentile of the Monte Carlo prediction is 0.0209, the 50$^{th}$ percentile is 0.0355, and the 90$^{th}$ percentile is 0.0619 mg/kg bw/day.

The Monte Carlo analysis revealed that exposure of small piscivorus birds to mercury in BI AOC could range from a minimum of 0.0248 to a maximum of 0.379 mg/kg bw/day. The mean exposure is 0.0894 mg/kg bw/day and the median exposure is 0.0829 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0473 and 0.153 mg/kg bw/day. Figure H3-15 shows the cumulative distribution of mercury intake rates for small piscivorus bird species in BI AOC.

Sensitivity analysis revealed that the FMR slope was the most important variable [Pearson correlation coefficient ($r_p$) = 0.63], followed by gross energy of fish ($r_p$ = -0.53) and the FMR power term ($r_p$ = 0.38).

The probability bounds estimated for small piscivorus birds are depicted in Figure H3-15. The 10$^{th}$ percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0316 and 0.116 mg/kg bw/day. The 50$^{th}$ percentile ranges between 0.0544 and 0.189 mg/kg bw/day, and the 90$^{th}$ percentile ranges between 0.0867 and 0.347 mg/kg bw/day. In comparison, the 10$^{th}$ percentile of the Monte

Carlo prediction is 0.0531, the 50th percentile is 0.0824, and the 90th percentile is 0.132 mg/kg bw/day.

***Mercury – Upper Calcasieu River AOC***

The Monte Carlo analysis revealed that exposure of piscivorus birds to mercury in the UCR AOC could range from a minimum of 0.00220 to a maximum of 0.0521 mg/kg bw/day. The mean exposure is 0.0115 mg/kg bw/day and the median exposure is 0.0104 mg/kg bw/day. Ninety percent of exposure estimates are between of 0.00536 and 0.0214 mg/kg bw/day. Figure H3-16 depicts the cumulative distribution of mercury intake rates for the average-sized piscivorus bird species.

Sensitivity analysis revealed that FMR power term was the most important variable [Pearson correlation coefficient ($r_p$) = 0.61] followed by FMR slope ($r_p$ = 0.53) and gross energy of fish ($r_p$ = -0.44).

The probability bounds estimated for average-sized piscivorus birds are depicted in Figure H3-16. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.00353 and 0.0148 mg/kg bw/day. The 50th percentile ranges between 0.00668 and 0.0255 mg/kg bw/day, and the 90th percentile ranges between 0.0133 and 0.0514 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo prediction is 0.00613, the 50th percentile is 0.0104, and the 90th percentile is 0.0181 mg/kg bw/day.

The Monte Carlo analysis revealed that exposure of small piscivorus birds to mercury in the UCR AOC could range from a minimum of 0.00697 to a maximum of 0.114 mg/kg bw/day. The mean exposure is 0.0261 mg/kg bw/day and the median exposure is 0.0242 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0139

and 0.0448 mg/kg bw/day. Figure H3-17 shows the cumulative distribution of mercury intake rates for the small piscivorus bird species in UCR AOC.

Sensitivity analysis revealed that the FMR slope was the most important variable [Pearson correlation coefficient ($r_p$) = 0.63], followed by gross energy of fish ($r_p$ = -0.53) and the FMR power term ($r_p$ = 0.38).

The probability bounds estimated for small piscivorus birds are depicted in Figure H3-17. The 10$^{th}$ percentile of the probability envelope formed by the lower and upper bounds ranges between 0.00901 and 0.0363 mg/kg bw/day. The 50$^{th}$ percentile ranges between 0.0157 and 0.0587 mg/kg bw/day, and the 90$^{th}$ percentile ranges between 0.0249 and 0.110 mg/kg bw/day. In comparison, the 10$^{th}$ percentile of the Monte Carlo prediction is 0.0155, the 50$^{th}$ percentile is 0.0240, and the 90$^{th}$ percentile is 0.0386 mg/kg bw/day.

***Mercury – Middle Calcasieu River AOC***

The Monte Carlo analysis revealed that exposure of average-sized piscivorus birds to mercury in the MCR AOC could range from a minimum of 0.00250 to a maximum of 0.0570 mg/kg bw/day. The mean exposure is 0.0126 mg/kg bw/day and the median exposure is 0.0114 mg/kg bw/day. Ninety percent of exposure estimates are between of 0.00586 and 0.0233 mg/kg bw/day. Figure H3-18 depicts the cumulative distribution of mercury intake rates for the average-sized piscivorus bird species.

Sensitivity analysis revealed that FMR power term was the most important variable [Pearson correlation coefficient ($r_p$) = 0.61] followed by FMR slope ($r_p$ = 0.53) and gross energy of fish ($r_p$ = -0.44).

The probability bounds estimated for average-sized piscivorus birds are depicted in Figure H3-18. The 10$^{th}$ percentile of the probability envelope formed by the lower and upper bounds ranges between 0.00402 and 0.0165 mg/kg bw/day. The 50$^{th}$ percentile ranges between 0.00758 and 0.0285 mg/kg bw/day, and the 90$^{th}$ percentile ranges between 0.0129 and 0.0574 mg/kg bw/day. In comparison, the 10$^{th}$ percentile of the Monte Carlo prediction is 0.00674, the 50$^{th}$ percentile is 0.0114, and the 90$^{th}$ percentile is 0.0198 mg/kg bw/day.

The Monte Carlo analysis revealed that exposure of small piscivorus birds to mercury in the MCR AOC could range from a minimum of 0.00755 to a maximum of 0.127 mg/kg bw/day. The mean exposure is 0.0286 mg/kg bw/day and the median exposure is 0.0266 mg/kg bw/day. Ninety percent of exposure estimates are between 0.0152 and 0.0489 mg/kg bw/day. Figure H3-19 shows the cumulative distribution of mercury intake rates for the small piscivorus bird species in MCR AOC.

Sensitivity analysis revealed that the FMR slope was the most important variable [Pearson correlation coefficient ($r_p$) = 0.63], followed by gross energy of fish ($r_p$ = -0.53) and the FMR power term ($r_p$ = 0.38).

The probability bounds estimated for small piscivorus birds are depicted in Figure H3-19. The 10$^{th}$ percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0102 and 0.0406 mg/kg bw/day. The 50$^{th}$ percentile ranges between 0.0178 and 0.0657 mg/kg bw/day, and the 90$^{th}$ percentile ranges between 0.0282 and 0.122 mg/kg bw/day. In comparison, the 10$^{th}$ percentile of the Monte Carlo prediction is 0.0172, the 50$^{th}$ percentile is 0.0265, and the 90$^{th}$ percentile is 0.0424 mg/kg bw/day.

***Mercury – Reference Areas***

The Monte Carlo analysis revealed that exposure of average-sized piscivorus birds to mercury in the reference areas could range from a minimum of 0.00116 to a maximum of 0.0260 mg/kg bw/day. The mean exposure is 0.00587 mg/kg bw/day and the median exposure is 0.00532 mg/kg bw/day. Ninety percent of exposure estimates are between 0.00274 and 0.0109 mg/kg bw/day. Figure H3-20 depicts the cumulative distribution of mercury intake rates for the average-sized piscivorus bird species.

Sensitivity analysis revealed that the FMR power term was the most important variable [Pearson correlation coefficient ($r_p$) = 0.61] followed by FMR slope ($r_p$ = 0.53) and gross energy of fish ($r_p$ = -0.44).

The probability bounds estimated for average-sized piscivorus birds are depicted in Figure H3-20. The 10$^{th}$ percentile of the probability envelope formed by the lower and upper bounds ranges between 0.00178 and 0.00799 mg/kg bw/day. The 50$^{th}$ percentile ranges between 0.00342 and 0.0137 mg/kg bw/day, and the 90$^{th}$ percentile ranges between 0.00576 and 0.0280 mg/kg bw/day. In comparison, the 10$^{th}$ percentile of the Monte Carlo prediction is 0.00311, the 50$^{th}$ percentile is 0.00529, and the 90$^{th}$ percentile is 0.00923 mg/kg bw/day.

The Monte Carlo analysis revealed that exposure of small piscivorus birds to mercury in the reference areas could range from a minimum of 0.00368 to a maximum of 0.0583 mg/kg bw/day. The mean exposure is 0.0133 mg/kg bw/day and the median exposure is 0.0124 mg/kg bw/day. Ninety percent of exposure estimates are between 0.00710 and 0.0229 mg/kg bw/day. Figure H3-21 shows the cumulative distribution of mercury intake rates for the small piscivorus bird species in the reference areas.

Sensitivity analysis revealed that the FMR slope was the most important variable [Pearson correlation coefficient ($r_p$) = 0.63], followed by gross energy of fish ($r_p$ = -0.53) and the FMR power term ($r_p$ = 0.38).

The probability bounds estimated for small piscivorus birds are depicted in Figure H3-21. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.00458 and 0.0196 mg/kg bw/day. The 50th percentile ranges between 0.00806 and 0.0315 mg/kg bw/day, and the 90th percentile ranges between 0.0127 and 0.0598 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo prediction is 0.00800, the 50th percentile is 0.0124, and the 90th percentile is 0.0197 mg/kg bw/day.

***Total PCBs – Bayou d'Inde AOC***

The Monte Carlo analysis revealed that exposure of piscivorus birds to total PCBs in BI AOC could range from a minimum of 0.00847 to a maximum of 0.179 mg/kg bw/day. The mean exposure is 0.0418 mg/kg bw/day and the median exposure is 0.0377 mg/kg bw/day. Ninety percent of exposure estimates are between of 0.0193 and 0.0783 mg/kg bw/day. Figure H3-22 depicts the cumulative distribution of total PCB intake rates for the average-sized piscivorus bird species.

Sensitivity analysis revealed that the FMR power term was the most important variable [Pearson correlation coefficient ($r_p$) = 0.60] followed by FMR slope ($r_p$ = 0.53) and gross energy of fish ($r_p$ = -0.44).

The probability bounds estimated for average-sized piscivorus birds are depicted in Figure H3-22. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0112 and 0.0549 mg/kg bw/day. The 50th percentile ranges between 0.0217 and 0.0936 mg/kg bw/day, and the 90th percentile

ranges between 0.0363 and 0.195 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo prediction is 0.0220, the 50th percentile is 0.0375, and the 90th percentile is 0.0665 mg/kg bw/day.

The Monte Carlo analysis revealed that exposure of small piscivorus birds to total PCBs in BI AOC could range from a minimum of 0.0247 to a maximum of 0.451 mg/kg bw/day. The mean exposure is 0.0950 mg/kg bw/day and the median exposure is 0.0879 mg/kg bw/day. Ninety percent of exposure estimates are between of 0.0499 and 0.164 mg/kg bw/day. Figure H3-23 depicts the cumulative distribution of total PCB intake rates for the small piscivorus bird species.

Sensitivity analysis revealed that the FMR slope was the most important variable [Pearson correlation coefficient ($r_p$) = 0.62] followed by gross energy of fish ($r_p$ = -0.52) and the FMR power term ($r_p$ = 0.37).

The probability bounds estimated for small piscivorus birds are depicted in Figure H3-23. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.0287 and 0.134 mg/kg bw/day. The 50th percentile ranges between 0.0512 and 0.215 mg/kg bw/day, and the 90th percentile ranges between 0.0800 and 0.414 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo prediction is 0.0561, the 50th percentile is 0.0876, and the 90th percentile is 0.142 mg/kg bw/day.

***Total PCBs – Reference Areas***

The Monte Carlo analysis revealed that exposure of average-sized piscivorus birds to total PCBs in the reference areas could range from a minimum of 0.00124 to a maximum of 0.0295 mg/kg bw/day. The mean exposure is 0.00600 mg/kg bw/day and the median exposure is 0.00544 mg/kg bw/day. Ninety percent of exposure

estimates are between of 0.00276 and 0.0113 mg/kg bw/day. Figure H3-24 depicts the cumulative distribution of PCB intake rates for the average-sized piscivorus bird species.

Sensitivity analysis revealed that the FMR power term was the most important variable [Pearson correlation coefficient ($r_p$) = 0.60] followed by FMR slope ($r_p$ = 0.53) and gross energy ($r_p$ = -0.44).

The probability bounds estimated for average-sized piscivorus birds are depicted in Figure H3-24. The 10$^{th}$ percentile of the probability envelope formed by the lower and upper bounds ranges between 0.00138 and 0.00963 mg/kg bw/day. The 50$^{th}$ percentile ranges between 0.00285 and 0.0160 mg/kg bw/day, and the 90$^{th}$ percentile ranges between 0.00464 and 0.0359 mg/kg bw/day. In comparison, the 10$^{th}$ percentile of the Monte Carlo prediction is 0.00318, the 50$^{th}$ percentile is 0.00540, and the 90$^{th}$ percentile is 0.00950 mg/kg bw/day.

The Monte Carlo analysis revealed that exposure of small piscivorus birds to PCBs in the reference areas could range from a minimum of 0.00350 to a maximum of 0.0644 mg/kg bw/day. The mean exposure is 0.0136 mg/kg bw/day and the median exposure is 0.0126 mg/kg bw/day. Ninety percent of exposure estimates are between of 0.00715 and 0.0235 mg/kg bw/day. Figure H3-25 depicts the cumulative distribution of PCB intake rates for the small piscivorus bird species.

Sensitivity analysis revealed that the FMR slope was the most important variable [Pearson correlation coefficient ($r_p$) = 0.62] followed by gross energy of fish ($r_p$ = -0.52) and the FMR power term ($r_p$ = 0.37).

The probability bounds estimated for small piscivorus birds are depicted in Figure H3-25. The 10th percentile of the probability envelope formed by the lower and upper bounds ranges between 0.00356 and 0.0234 mg/kg bw/day. The 50th percentile ranges between 0.00680 and 0.0365 mg/kg bw/day, and the 90th percentile ranges between 0.0103 and 0.0756 mg/kg bw/day. In comparison, the 10th percentile of the Monte Carlo prediction is 0.00802, the 50th percentile is 0.0126, and the 90th percentile is 0.0203 mg/kg bw/day.

## 3.2 Review of Historical Data

Levels of Aroclor 1254 in tissues of fish collected from CH2M Hill's Calcasieu Estuary Biological Monitoring Program were consistent with levels found in the Phase II Sampling Program. Levels in whole body determined in 2001 during Phase II Sampling and levels in fillet recorded since 1991 by CH2M Hill were used for statistical analysis. For comparison, fillet concentrations were estimated for the samples collected from the Phase II Sampling Program using the following equation:

$$C_f = C_{wb} / 2.3 \qquad \textbf{EQUATION \#8}$$

where $C_{wb}$ is whole-body concentration and $C_f$ is fillet concentration (SAIC 1993).

Annual geometric mean concentrations in fillet of red drum, black drum, spotted seatrout, sand seatrout and southern flounder were calculated for the four AOCs. The geometric mean concentration of Aroclor 1254 in fillet collected from the BI AOC during the Phase II Sampling Program was 0.016 mg/kg, with minimum and maximum concentrations of 0.002 mg/kg and 0.230 mg/kg, respectively. Since 1991, the annual geometric mean concentrations determined by CH2M Hill's Biological

Monitoring Program ranged from 0.028 mg/kg to 0.133 mg/kg and the minimum and maximum concentrations were 0.003 mg/kg and 1.080 mg/kg, respectively (Figure H3-26).

The geometric mean concentration of Aroclor 1254 in fillet collected from the UCR AOC during the Phase II Sampling Program was 0.013 mg/kg, with minimum and maximum concentrations of 0.002 mg/kg and 0.478 mg/kg, respectively. Since 1991, the annual geometric mean concentrations determined by CH2M Hill's Biological Monitoring Program ranged from 0.006 mg/kg to 0.040 mg/kg and the minimum and maximum concentrations were 0.005 mg/kg and 0.232 mg/kg, respectively (Figure H3-27).

The geometric mean concentration of Aroclor 1254 in fillet collected from the MCR AOC during the Phase II Sampling Program was 0.013 mg/kg, with a minimum and maximum concentration of 0.002 mg/kg and 0.317 mg/kg, respectively. Since 1991, the annual geometric mean concentrations determined by CH2M Hill's Biological Monitoring Program ranged from 0.008 mg/kg to 0.031 mg/kg and the minimum and maximum concentrations were 0.003 mg/kg and 0.221 mg/kg, respectively (Figure H3-28).

The geometric mean concentration of Aroclor 1254 in fillet collected from the Calcasieu Estuary reference areas during the Phase II Sampling Program was 0.006 mg/kg, with a minimum and maximum concentration of 0.002 mg/kg and 0.029 mg/kg, respectively. Since 1991, the annual geometric mean concentrations determined by CH2M Hill's Biological Monitoring Program ranged from 0.006 mg/kg to 0.016 mg/kg and the minimum and maximum concentrations were 0.003 mg/kg and 0.378 mg/kg, respectively (Figure H3-29).

The comparison of historical data sets between the Phase II Sampling Program and CH2M Hill's Biological Monitoring Program showed that there was less than one order of magnitude difference in levels of total PCBs in fish tissues between the ten years of historical data and data collected in the Phase II Sampling Program. In most cases, the difference was less than four fold. This demonstrates that the results of the ecological risk assessment for piscivorus birds using data from the Phase II Sampling Program are likely to be temporally representative.

## 3.3 Risk Assessment

For each exposure scenario, a low, indeterminate, and high category of risk was determined for piscivorus birds. These categories of risk were derived using the following guidance:

1. If the probability of exceeding the lower toxicity threshold is less than 20%, the risk to piscivorus birds is considered low;
2. If the probability of exceeding the upper toxicity threshold is greater than 20%, the risk to piscivorus birds is considered high; and,
3. All other probabilities are considered to have indeterminate risk.

***TCDD-TEQs – Bayou d'Inde AOC***

The Monte Carlo predictions for total daily intake of TCDD-TEQs by average-sized piscivorus birds indicate there is a 67% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. The lower probability bound suggests a 24% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. The upper probability bound suggests a 100% likelihood of exceeding the lower toxicity threshold and a 1% probability of exceeding the upper toxicity threshold. Therefore,

TCDD-TEQs pose indeterminate risks to the survival and reproduction of average-sized piscivorus birds in BI AOC (Table H3-4).

Comparison of exposure to the Appendix G benchmark reveals there is a 0% probability the Monte Carlo-predicted exposure will exceed the benchmark. The probability bounds analysis indicates that the probability of exceeding the Appendix G benchmark could range from 0 to 57% (Table H3-5).

The Monte Carlo predictions for total daily intake of TCDD-TEQs by small piscivorus birds indicate a 99% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. The lower probability bound suggests an 89% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. The upper probability bound suggests a 100% probability of exceeding the lower toxicity threshold and a 1% probability of exceeding the upper toxicity threshold. Therefore, TCDD-TEQs pose indeterminate risks to the survival and reproduction of small piscivorus birds in BI AOC (Table H3-4).

Comparison of exposure to the Appendix G benchmark shows a 37% probability that the Monte Carlo-predicted exposure will exceed the benchmark. The probability bounds analysis indicates that the probability of exceeding the Appendix G benchmark could range from 3 to 100% (Table H3-5).

***TCDD-TEQs – Middle Calcasieu River AOC***

The Monte Carlo predictions for total daily intake of TCDD-TEQs by average-sized piscivorus birds indicate there is a 4% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. The lower probability bound is below the lower and upper toxicity thresholds. The upper

probability bound suggests a 100% likelihood of exceeding the lower toxicity threshold and a 1% probability of exceeding the upper toxicity threshold. Therefore, average-sized piscivorus birds exposed to TCDD-TEQs in the MCR AOC are at low risk of experiencing adverse effects to survival or reproduction (Table H3-4).

Comparison of exposure to the Appendix G benchmark reveals there is a 0% probability the Monte Carlo-predicted exposure will exceed the benchmark. The probability bounds analysis indicates that the probability of exceeding the Appendix G benchmark could range from 0 to 43% (Table H3-5).

The Monte Carlo predictions for total daily intake of TCDD-TEQs by small piscivorus birds indicate a 59% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. The lower probability bound suggests a 24% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. The upper probability bound suggests a 100% probability of exceeding the lower toxicity threshold and a 4% probability of exceeding the upper toxicity threshold. Therefore, TCDD-TEQs pose indeterminate risks to the survival and reproduction of small piscivorus birds in the MCR AOC (Table H3-4).

Comparison of exposure to the Appendix G benchmark shows a 0% probability that the Monte Carlo-predicted exposure will exceed the benchmark. The probability bounds analysis indicates that the probability of exceeding the Appendix G benchmark could range from 0 to 85% (Table H3-5).

***TCDD-TEQs – Reference Areas***

The Monte Carlo predictions for total daily intake of TCDD-TEQs by average-sized piscivorus birds indicate there is a 0% probability of exceeding the lower and upper

toxicity thresholds. The lower probability bound is below the lower and upper toxicity thresholds. The upper probability bound suggests a 100% likelihood of exceeding the lower toxicity threshold and a 1% probability of exceeding the upper toxicity threshold. Therefore, average-sized piscivorus birds exposed to TCDD-TEQs in the reference areas are at low risk of experiencing adverse effects to survival or reproduction (Table H3-4).

Comparison of exposure to the Appendix G benchmark reveals a 0% probability that the Monte Carlo-predicted exposure and lower probability bound will exceed the benchmark. The probability bounds analysis indicates that the probability of exceeding the Appendix G benchmark could range from 0 to 42% (Table H3-5).

The Monte Carlo predictions for total daily intake of TCDD-TEQs by small piscivorus birds indicate there is a 34% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. The lower probability bound suggests a 22% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. The upper probability bound suggests a 100% probability of exceeding the lower toxicity threshold and a 4% probability of exceeding the upper toxicity threshold. Therefore, TCDD-TEQs pose indeterminate risks to the survival and reproduction of small piscivorus birds in the reference areas (Table H3-4).

Comparison of exposure to the Appendix G benchmark reveals a 0% probability the Monte Carlo-predicted exposure will exceed the benchmark. The probability bounds analysis indicates that the probability of exceeding the Appendix G benchmark could range from 0 to 85% (Table H3-5).

***Selenium – Bayou d'Inde AOC***

The Monte Carlo predictions for total daily intake of selenium by average-sized piscivorus birds indicate a 7% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. The lower probability bound is below the lower and upper toxicity thresholds. The upper probability bound suggests a 64% probability of exceeding the lower toxicity threshold and a 7% probability of exceeding the upper toxicity threshold. Therefore, average-sized piscivorus birds exposed to selenium in BI AOC are at a low risk of experiencing adverse effects to survival or reproduction (Table H3-4).

Comparison of exposure to the Appendix G benchmark reveals a 0% probability the Monte Carlo-predicted exposure or lower probability bound will exceed the benchmark. The probability bounds analysis indicates that the probability of exceeding the Appendix G benchmark could range from 0 to 3% (Table H3-5).

The Monte Carlo predictions for total daily intake of selenium by small piscivorus birds indicate a 72% probability of exceeding the lower toxicity threshold and a 2% probability of exceeding the upper toxicity threshold. The lower probability bound suggests that there is a 36% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. The upper probability bound suggests a 100% probability of exceeding the lower toxicity threshold and a 55% probability of exceeding the upper toxicity threshold. Therefore, selenium poses indeterminate risks to the survival and reproduction of small piscivorus birds in BI AOC (Table H3-4).

Comparison of exposure to the Appendix G benchmark reveals a 0% probability of the Monte Carlo-predicted exposure exceeding the benchmark. The probability

bounds analysis indicates that the probability of exceeding the Appendix G benchmark could range from 0 to 33% (Table H3-5).

***Selenium – Reference Areas***

The Monte Carlo predictions for total daily intake of selenium by average-sized piscivorus birds indicate a 6% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. The lower probability bound is below the lower and upper toxicity thresholds. The upper probability bound suggests a 75% probability of exceeding the lower toxicity threshold and a 12% probability of exceeding the upper toxicity threshold. Therefore, average-sized piscivorus birds exposed to selenium in the reference areas are at a low risk of experiencing adverse effects to survival or reproduction (Table H3-4).

Comparison of exposure to the Appendix G benchmark reveals a 0% probability that the Monte Carlo-predicted exposure will exceed the benchmark. The probability bounds analysis indicates that the probability of exceeding the Appendix G benchmark could range from 0 to 6% (Table H3-5).

The Monte Carlo predictions for total daily intake of selenium by small piscivorus birds indicate a 68% probability of exceeding the lower toxicity threshold and a 2% probability of exceeding the upper toxicity threshold. The lower probability bound suggests a 20% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. The upper probability bound suggests a 100% probability of exceeding the lower toxicity threshold and a 67% probability of exceeding the upper toxicity threshold. Therefore, selenium poses indeterminate risks to the survival and reproduction of small piscivorus birds in the reference areas (Table H3-4).

Comparison of exposure to the Appendix G benchmark reveals a 0% probability of the Monte Carlo-predicted exposure exceeding the benchmark. The probability bounds analysis indicates that the probability of exceeding the Appendix G benchmark could range from 0 to 44% (Table H3-5).

***Mercury – Bayou d'Inde AOC***

The Monte Carlo predictions for total daily intake of mercury by average-sized piscivorus birds indicate a 9% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. The lower probability bound is below the lower and upper toxicity thresholds. The upper probability bound suggests a 70% probability of exceeding the lower toxicity threshold and a 1% probability of exceeding the upper toxicity threshold. Therefore, average-sized piscivorus birds exposed to mercury in BI AOC are at a low risk of experiencing adverse effects to survival or reproduction (Table H3-4).

Comparison of exposure to the Appendix G benchmark reveals a 91% probability the Monte Carlo-predicted exposure will exceed the benchmark. The probability bounds analysis indicates that the probability of exceeding the Appendix G benchmark could range from 61 to 100% (Table H3-5).

The Monte Carlo predictions for total daily intake of mercury by small piscivorus birds indicate a 77% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. The lower probability bound suggests a 34% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. The upper probability bound suggests a 100% probability of exceeding the lower toxicity threshold and a 2% probability of exceeding the upper toxicity threshold. Therefore, mercury poses

indeterminate risks to the survival and reproduction of small piscivorus birds in BI AOC (Table H3-4).

Comparison of exposure to the Appendix G benchmark reveals a 100% probability that the Monte Carlo-predicted exposure will exceed the benchmark. The probability bounds analysis indicates that the probability of exceeding the Appendix G benchmark could range from 98 to 100% (Table H3-5).

***Mercury – Upper Calcasieu River AOC***

The Monte Carlo predictions for total daily intake of mercury by average-sized piscivorus birds indicate a 0% probability of exceeding the lower and upper toxicity thresholds. The lower probability bound is below the lower and upper toxicity thresholds. The upper probability bound suggests a 5% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. Therefore, average-sized piscivorus birds exposed to mercury in the UCR AOC are at low risk of experiencing adverse effects to survival or reproduction (Table H3-4).

Comparison of exposure to the Appendix G benchmark reveals a 6% probability the Monte Carlo-predicted exposure will exceed the benchmark. The probability bounds analysis indicates that the probability of exceeding the Appendix G benchmark could range from 0 to 69% (Table H3-5).

The Monte Carlo predictions for total daily intake of mercury by small piscivorus birds indicates a 0% probability of exceeding the lower and upper toxicity thresholds. The lower probability bound is below the lower and upper toxicity thresholds. The upper probability bound suggests a 42% probability of exceeding the lower toxicity threshold and a 1% probability of exceeding the upper toxicity threshold. Therefore,

small piscivorus birds exposed to mercury in the UCR AOC are at a low risk of experiencing adverse effects to survival or reproduction (Table H3-4).

Comparison of exposure to the Appendix G benchmark reveals a 69% probability the Monte Carlo-predicted exposure will exceed the benchmark. The probability bounds analysis indicates that the probability of exceeding the Appendix G benchmark could range from 25 to 100% (Table H3-5).

***Mercury – Middle Calcasieu River AOC***

The Monte Carlo predictions for total daily intake of mercury by average-sized piscivorus birds indicate a 0% probability of exceeding the lower and upper toxicity thresholds. The lower probability bound is below the lower and upper toxicity thresholds. The upper probability bound suggests a 7% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. Therefore, average-sized piscivorus birds exposed to mercury in the MCR AOC are at a low risk of experiencing adverse effects to survival or reproduction (Table H3-4).

Comparison of exposure to the Appendix G benchmark reveals a 9% probability the Monte Carlo-predicted exposure will exceed the benchmark. The probability bounds analysis indicates that the probability of exceeding the Appendix G benchmark could range from 0 to 78% (Table H3-5).

The Monte Carlo predictions for total daily intake of mercury by small piscivorus birds indicate a 0% probability of exceeding the lower and upper toxicity thresholds. The lower probability bound is below the lower and upper toxicity thresholds. The upper probability bound suggests a 53% probability of exceeding the lower toxicity threshold and a 1% probability of exceeding the upper toxicity threshold. Therefore,

small piscivorus birds exposed to mercury in the MCR AOC are at a low risk of experiencing adverse effects to survival or reproduction (Table H3-4).

Comparison of exposure to the Appendix G benchmark reveals a 78% probability the Monte Carlo-predicted exposure will exceed the benchmark. The probability bounds analysis indicates that the probability of exceeding the Appendix G benchmark could range from 37 to 100% (Table H3-5).

***Mercury – Reference Areas***

The Monte Carlo and probability bounds predictions for total daily intake rates of mercury by average-sized piscivorus birds indicate a 0% probability of the lower or upper toxicity thresholds being exceeded. There is a 0% probability of the lower probability bound exceeding the lower and upper toxicity thresholds. The upper probability bound suggests a 2% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. Therefore, average-sized piscivorus exposed to mercury in the reference areas are at a low risk of experiencing adverse effects to survival or reproduction (Table H3-4).

Comparison of exposure to the Appendix G benchmark reveals a 0% probability the Monte Carlo-predicted exposure will exceed the benchmark. The probability bounds analysis indicates that the probability of exceeding the Appendix G benchmark could range from 0 to 23% (Table H3-5).

The Monte Carlo and probability bounds predictions for total daily intake rates of mercury by small piscivorus birds indicate a 0% probability of the lower or upper toxicity thresholds being exceeded. There is a 0% probability of the lower probability bound exceeding the lower or upper toxicity threshold. The upper probability bound suggests an 8% probability of exceeding the lower toxicity threshold and a 0%

probability of exceeding the upper toxicity threshold. Therefore, small piscivorus birds exposed to mercury in the reference areas are at a low risk of experiencing adverse effects to survival or reproduction (Table H3-4).

Comparison of exposure to the Appendix G benchmark reveals a 9% probability the Monte Carlo-predicted exposure will exceed the benchmark. The probability bounds analysis indicates that the probability of exceeding the Appendix G benchmark could range from 0 to 88% (Table H3-5).

***Total PCBs – Bayou d'Inde AOC***

The Monte Carlo predictions for total daily intake of total PCBs by average-sized piscivorus birds indicate a 0% probability of exceeding the lower and upper toxicity thresholds. The lower probability bound is below the lower and upper toxicity thresholds. The upper probability bound suggests a 32% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. Therefore, average-sized piscivorus birds exposed to total PCBs in BI AOC are at a low risk of experiencing adverse effects to survival or reproduction (Table H3-4).

Comparison of exposure to the Appendix G benchmark reveals a 0% probability the Monte Carlo-predicted exposure or lower probability bound will exceed the benchmark. The probability bounds analysis indicates that the probability of exceeding the Appendix G benchmark could range from 0 to 1% (Table H3-5).

The Monte Carlo predictions for total daily intake of total PCBs by small piscivorus birds indicate a 20% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. The lower probability bound is below the lower and upper toxicity thresholds. The upper probability bound

suggests a 96% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. Therefore, total PCBs pose indeterminate risks to the survival and reproduction of small piscivorus birds in BI AOC (Table H3-4).

Comparison of exposure to the Appendix G benchmark reveals a 0% probability the Monte Carlo-predicted exposure will exceed the benchmark. The probability bounds analysis indicates that the probability of exceeding the Appendix G benchmark could range from 0 to 3% (Table H3-5).

***Total PCBs – Reference Areas***

The Monte Carlo and probability bounds predictions for total daily intake rates of total PCBs by average-sized piscivorus birds indicate a 0% probability of the lower or upper toxicity thresholds being exceeded. The lower probability bound is below the lower and upper toxicity thresholds. The upper probability bound suggests a 1% probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper toxicity threshold. Therefore, average-sized piscivorus birds exposed to total PCBs in the reference areas are at a low risk of experiencing adverse effects to survival or reproduction (Table H3-4).

Comparison of exposure to the Appendix G benchmark reveals a 0% probability that the Monte Carlo-predicted exposure, lower or upper probability bounds will exceed the benchmark (Table H3-5).

The Monte Carlo and lower probability bounds predictions for total daily intake rates of total PCBs by small piscivorus birds indicate a 0% probability of the lower or upper toxicity thresholds being exceeded. The lower probability bound is below the lower and upper toxicity thresholds. The upper probability bound suggests a 3%

probability of exceeding the lower toxicity threshold and a 0% probability of exceeding the upper probability bound. Therefore, small piscivorus birds exposed to total PCBs in the reference areas are at a low risk of experiencing adverse effects to survival or reproduction (Table H3-4).

Comparison of exposure to the Appendix G benchmark reveals a 0% probability of the Monte Carlo-predicted exposure, or lower or upper probability bounds exceeding the benchmark (Table H3-5).

# 4.0 Uncertainty Analysis

There are a number of sources of uncertainty in the assessments of risk to piscivorus birds, including uncertainties in the conceptual model, and in the exposure, effects and risk assessments. As each of these sources of uncertainty can influence the estimations of risk, it is important to describe and, when possible, quantify the magnitude and direction of such uncertainties. In this way, it is possible to evaluate the level of confidence that can be placed in the assessments conducted. The uncertainties associated with the assessment of risks to piscivorus birds are described in the following sections.

**Uncertainties Associated With the Conceptual Model** - The conceptual model is intended to define the linkages between stressors, potential exposure, and predicted effects on ecological receptors. As such, the conceptual model provides the scientific basis for selecting assessment and measurement endpoints to support the risk assessment process. Potential uncertainties arise from lack of knowledge regarding ecosystem functions, failure to adequately address spatial and temporal variability in the evaluations of sources, fate, and effects, omission of stressors, and overlooking

secondary effects (USEPA 1998). The types of uncertainties that are associated with the conceptual model that links substance sources to effects on piscivorus birds include those associated with the identification of COCs, environmental fate and transport of COCs, exposure pathways, receptors at risk, and ecological effects. Of these, the identification of exposure pathways probably represents the primary source of uncertainty in the conceptual model. In this assessment, it was assumed that exposure to contaminated food represents the most important pathway for exposing piscivorus birds to COCs. Although unlikely to be important, other pathways could contribute to exposure and perhaps increase risk somewhat.

**Uncertainties Associated With the Exposure Assessment** - The exposure assessment is intended to describe the actual or potential co-occurrence of stressors with receptors. As such, the exposure assessment identifies the exposure pathways and the intensity and extent of contact with stressors for each receptor or group of receptors at risk. There are a number of potential sources of uncertainty in the exposure assessment, including measurement errors, extrapolation errors, and data gaps.

In this assessment, tissue residues in fish was the measurement endpoint used to evaluate exposure of piscivorus birds to COCs. Analytical errors and descriptive errors represent potential sources of uncertainty in this measurement.

Three approaches were used to address concerns relative to these sources of uncertainty.

First, analytical errors were evaluated using information on the accuracy, precision, and detection limits (DL) generated to support the Phase I and Phase II sampling programs. The results of this analysis indicated that most of the data used in this

assessment met the project data quality objectives (see Appendix B1 for more details). Second, all data entry, data translation, and data manipulations were audited to ensure their accuracy. Data auditing involved 10% number-for-number checks against the primary data source initially, increasing to 100% number-for-number checks if significant errors were detected in the initial auditing step. Finally, statistical analyses of data were conducted to evaluate data distributions, identify appropriate summary statistics, and evaluate variability in the observations. Using these techniques, we were able to identify outliers and, if the outlier was due to an error, correct the outlier values.

According to the Monte Carlo sensitivity analyses, the FMR slope and power terms were among the most influential variables driving the predicted intake rates. Unfortunately, a precise estimate of the FMR was not possible, as measured metabolic rates for piscivorus birds used in this assessment were not available in the literature. Instead, the FMR for piscivorus birds was estimated using allometric equations. This introduced some degree of uncertainty into the exposure estimates because the allometric relationships were not only associated with some fitting error, but also were based on many bird species, some of which were very different from those represented here. However, given the lack of empirical data on species specific to the current assessment, it is difficult to judge the magnitude of the uncertainty introduced by the use of the allometric model rather than empirical data.

Other sensitive variables that influenced the exposure estimates included the gross energy of food and the food assimilation efficiency. These variables were also somewhat uncertain because no feeding studies were specifically performed in the Calcasieu Estuary on the species of interest. Rather, diet compositions were matched to those reported in the literature from birds raised in a laboratory or collected from other geographical locations. In consequence, the quantification of food gross energy

and assimilation efficiency was limited to the fish food group, without considering specific fish species. Some of the COC concentrations from the AOCs were derived from small sample sizes. This was a major source of uncertainty in the exposure assessment.

**Uncertainties in the Effects Assessment** - The effects assessment is intended to describe the effects caused by stressors, link them to the assessment endpoints, and evaluate how effects change with fluctuations in the levels (i.e., concentrations or doses) of the various stressors. There are several sources of uncertainty in the assessment of effects including measurement errors, extrapolation errors, model fit errors, and data gaps.

Insufficient data were available to create dose-response relationships for key species. As a result, toxicity threshold ranges were developed using surrogate species. This introduced uncertainty because it is not known whether laboratory raised and tested birds have the same sensitivities as birds living in the wild.

# 5.0 Conclusions

***TCDD-TEQs***

No areas in the Calacasieu Estuary contained TCDD-TEQs at levels representing a high risk to the survival or reproduction of piscivorus birds. For average-sized piscivorus birds, risks were indeterminate in BI AOC and low in the MCR AOC and reference areas. Risks were indeterminate for small piscivorus birds in BI AOC, MCR AOC, and the reference areas.

***Selenium***

No areas in the Calcasieu Estuary had selenium at levels representing a high risk to the survival or reproduction of piscivorus birds. Average-sized piscivorus birds in BI AOC and the reference areas were at low risk. The risks to small piscivorus birds were indeterminate in BI AOC and the reference areas.

***Mercury***

No areas in the Calcasieu Estuary contained mercury at levels representing a high risk to the survival or reproduction of piscivorus birds. Risk of adverse effects were low for average-sized piscivorus birds in BI AOC, MCR AOC, UCR AOC, and the reference areas. For small piscivorus birds, risks were indeterminate in BI AOC and low in MCR AOC, UCR AOC, and the reference areas.

***Total PCBs***

No areas in the Calcasieu Estuary had total PCBs at levels representing a high risk to the survival or reproduction of piscivorus birds. Risks were low for average-sized piscivorus birds in BI AOC and the reference areas. For small piscivorus birds, risks were indeterminate in BI AOC and low in the reference areas.

***Probabilistic Risk Assessment Limitations***

There are several limitations of the probabilistic risk analyses that influence our confidence regarding the above risk statements. These include:

- The sensitivity analyses for the Monte Carlo simulations indicated that the FMR slope and power terms were among the most important input variables for predicting intake rates. The FMR slope and power terms used in the analyses were based on the allometric equation from Nagy (1987). No corresponding measurements of this variable are available for

piscivorus bird species. The potential magnitude and direction of the uncertainty associated with lack of empirical data on metabolic rate are unknown. We did, however, investigate the possible consequences of the uncertainty in this variable due to model error (i.e., the error associated with the lack of fit of the allometric model that relates FMR to body weight). This source of uncertainty did not strongly impact our conclusions regarding risk;

- The analyses assumed that the hypothetical receptor forages exclusively in the Calcasieu Estuary and is a year round inhabitant. Some piscivorus bird species, however, have larger feeding ranges (e.g., osprey, brown pelicans) and are not year round inhabitants (e.g., belted kingfisher, osprey, terns). We would expect risks to these bird species to be lower than for the hypothetical receptors considered in our analyses;

- The effects analyses pointed out key sources of uncertainty. First, no data were available for piscivorus bird species. Second, differing environmental conditions between the laboratory and the field introduced uncertainty to the estimation of effects doses; and,

The above described limitations are common to wildlife risk assessments and indicate the value of having other lines of evidence to help characterize risks. Biological surveys and ambient toxicity testing are two such lines of evidence. No *in situ* or whole media feeding studies are available, however, for piscivorus birds in the Calcasieu Estuary. Formal biological surveys that relate degree of COC contamination to abundances of different piscivorus bird species have not been

conducted. However, bird banding and other surveys by the State of Louisiana indicate that many species of piscivorus birds (e.g., belted kingfishers, osprey, brown pelicans, terns and others) are common throughout the estuary.

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# Tables

**Table H3-1. Risk quotients for contaminants of concern.**

| Contaminant of Concern (COC) | Area | Risk Quotient | Proceed to Probabilistic Assessment? |
|---|---|---|---|
| ***TCDD – TEQs*** | Bayou d'Inde | 1.73 | Yes |
| | Upper Calcasieu River | 0.278 | No |
| | Middle Calcasieu River | 1.52 | Yes |
| | Reference Areas | 0.752 | Yes |
| ***Selenium*** | Bayou d'Inde | 1.22 | Yes |
| | Upper Calcasieu River | 0.839 | No |
| | Middle Calcasieu River | 0.924 | No |
| | Reference Areas | 0.605 | Yes |
| ***Mercury*** | Bayou d'Inde | 28.4 | Yes |
| | Upper Calcasieu River | 6.13 | Yes |
| | Middle Calcasieu River | 6.53 | Yes |
| | Reference Areas | 2.59 | Yes |
| ***Total PCBs*** | Bayou d'Inde | 1.03 | Yes |
| | Upper Calcasieu River | 0.038 | No |
| | Middle Calcasieu River | 0.142 | No |
| | Reference Areas | 0.580 | Yes |

TCDD = tetrachlorodibenzo-*p* -dioxin; TEQs = toxic equivalents; PCBs = polychlorinated biphenyls.

**Table H3-2. Monte Carlo analysis input variables.**

| Variable | | | Distribution | Parameters |
|---|---|---|---|---|
| Body Weight: average-sized species ($BW$; kg) | | | normal | Mean = 1.25, SD = 0.188 |
| Body Weight: small-sized species ($BW$; kg) | | | normal | Mean = 0.0431, SD = 0.00647 |
| Free Metabolic Rate: average-sized and small species ($FMR$; Kcal/kg bw/day) | | | $FMR = aBW^b$ | |
| a = FMR-slope | | | normal | Mean = 0.681, SD = 0.102 |
| b = FMR-power | | | normal | Mean = 0.749, SD = 0.037 |
| Gross Energy ($GE_f$; Kcal/kg) | | | normal | Mean = 1200, SD = 240 |
| Assimilation Efficiency ($AE_f$; Unitless) | | | beta | alpha = 15, beta = 4, scale = 1.0 |
| ***Contaminants of Concern (COCs) - Input for Monte Carlo*** | | | | |
| **COCs** | **Area** | **Tissue Classification** | | |
| TCDD – TEQs | Bayou d'Inde | $C_{fish}$ Classes $_{1, 2\ A\text{-}B, 3\ A\text{-}B}$ (ng/kg ww) | lognormal | Mean = 82.7, SD = 2.06 |
| | Middle Calcasieu River | $C_{fish}$ Classes $_{1, 2\ A\text{-}B, 3\ A\text{-}B}$ (ng/kg ww) | lognormal | Mean = 32.0, SD = 1.63 |
| | Reference Areas | $C_{fish}$ Classes $_{1, 2\ A\text{-}B, 3\ A\text{-}B}$ (ng/kg ww) | lognormal | Mean = 25.3, SD = 0.640 |
| Selenium | Bayou d'Inde | $C_{fish}$ Classes $_{1, 2\ A\text{-}B, 3\ A\text{-}B}$ (mg/kg ww) | lognormal | Mean = 0.552, SD = 0.00587 |
| | Reference Areas | $C_{fish}$ Classes $_{1, 2\ A\text{-}B, 3\ A\text{-}B}$ (mg/kg ww) | lognormal | Mean = 0.530, SD = 0.0130 |
| Mercury | Bayou d'Inde | $C_{fish}$ Classes $_{1, 2\ A\text{-}B, 3\ A\text{-}B}$ (mg/kg ww) | lognormal | Mean = 0.174, SD = 0.00469 |
| | Upper Calcasieu River | $C_{fish}$ Classes $_{1, 2\ A\text{-}B, 3\ A\text{-}B}$ (mg/kg ww) | lognormal | Mean = 0.0507, SD = 0.00117 |

**Table H3-2. Monte Carlo analysis input variables.**

| Variable | | | Distribution | Parameters |
|---|---|---|---|---|
| **COCs (cont.)** | **Area (cont.)** | **Tissue Classification (cont.)** | | |
| Mercury (cont.) | Middle Calcasieu River | $C_{fish}$ Classes $_{1, 2 A-B, 3 A-B}$ (mg/kg ww) | lognormal | Mean = 0.0556, SD = 0.000944 |
| | Reference Areas | $C_{fish}$ Classes $_{1, 2 A, 3 A-B}$ (mg/kg ww) | lognormal | Mean = 0.0259, SD = 0.000408 |
| Total PCBs | Bayou d'Inde | $C_{fish}$ Classes $_{1, 2 A-B, 3 A-B}$ (mg/kg ww) | lognormal | Mean = 0.184, SD = 0.0115 |
| | Reference Areas | $C_{fish}$ Classes $_{1, 2 A, 3 A-B}$ (mg/kg ww) | lognormal | Mean = 0.0265, SD = 0.00181 |

SD - Standard deviation; TCDD = tetrachlorodibenzo-*p* -dioxin; TEQs = toxic equivalents; PCBs = polychlorinated biphenyls.

**Table H3-3. Probability Bounds analysis input variables.**

| Variable | | | Distribution | Parameters |
|---|---|---|---|---|
| ***Probability Bounds*** | | | | |
| Body Weight: average-sized species (*BW*; kg) | | | normal | Mean = 1.25, SD = 0.188 |
| Body Weight: small-sized species (*BW*; kg) | | | normal | Mean = 0.0431, SD = 0.00647 |
| Free Metabolic Rate: average-sized species (FMR; Kcal/kg bw/day) | | | $FMR = aBW^b$ | |
| a = FMR-slope | | | normal | Mean = 0.681, SD = 0.102 |
| b = FMR-power | | | normal | Mean = 0.749, SD = 0.037 |
| Gross Energy - Fish ($GE_f$; Kcal/kg) | | | lognormal | Mean = 1200; SD = 240 |
| Assimilation Efficiency ($AE_f$, unitless) | | | minmaxmean | 0.44, 0.99, 0.79 |
| ***Contaminants of Concern (COCs) - Input for Probability Bounds*** | | | | |
| **COCs** | **Area** | **Tissue Classification** | | |
| TCDD – TEQs | Bayou d'Inde | $C_{fish}$ Classes $_{1, 2\ A\text{-}B, 3\ A\text{-}B}$ (ng/kg ww) | lognormal | Mean = 93.1, SD = 15.8 |
| | Middle Calcasieu River | $C_{fish}$ Classes $_{1, 2\ A\text{-}B, 3\ A\text{-}B}$ (ng/kg ww) | lognormal | Mean = 59.5, SD = 44.6 |
| | Reference Areas | $C_{fish}$ Classes $_{1, 2\ A\text{-}B, 3\ A\text{-}B}$ (ng/kg ww) | lognormal | Mean = 58.0, SD = 44.6 |
| Selenium | Bayou d'Inde | $C_{fish}$ Classes $_{1, 2\ A\text{-}B, 3\ A\text{-}B}$ (mg/kg ww) | lognormal | Mean = 0.583, SD = 0.0346 |
| | Reference Areas | $C_{fish}$ Classes $_{1, 2\ A\text{-}B, 3\ A\text{-}B}$ (mg/kg ww) | lognormal | Mean = 0.574, SD = 0.0857 |
| Mercury | Bayou d'Inde | $C_{fish}$ Classes $_{1, 2\ A\text{-}B, 3\ A\text{-}B}$ (mg/kg ww) | lognormal | Mean = 0.179, SD = 0.0154 |
| | Upper Calcasieu River | $C_{fish}$ Classes $_{1, 2\ A\text{-}B, 3\ A\text{-}B}$ (mg/kg ww) | lognormal | Mean = 0.0537, SD = 0.00576 |

**Table H3-3. Probability Bounds analysis input variables.**

| Variable | | | Distribution | Parameters |
|---|---|---|---|---|
| **COCs (cont.)** | **Area (cont.)** | **Tissue Classification (cont.)** | | |
| Mercury (cont.) | Middle Calcasieu River | $C_{fish}$ Classes $_{1, 2\ A\text{-}B, 3\ A\text{-}B}$ (mg/kg ww) | lognormal | Mean = 0.0605, SD = 0.00625 |
| | Reference Areas | $C_{fish}$ Classes $_{1, 2\ A, 3\ A\text{-}B}$ (mg/kg ww) | lognormal | Mean = 0.0282, SD = 0.00345 |
| Total PCBs | Bayou d'Inde | $C_{fish}$ Classes $_{1, 2\ A\text{-}B, 3\ A\text{-}B}$ (mg/kg ww) | lognormal | Mean = 0.186, SD = 0.0271 |
| | Reference Areas | $C_{fish}$ Classes $_{1, 2\ A, 3\ A\text{-}B}$ (mg/kg ww) | lognormal | Mean = 0.0284, SD = 0.00700 |

SD - Standard deviation; TCDD = tetrachlorodibenzo-*p*-dioxin; TEQs = toxic equivalents; PCBs = polychlorinated biphenyls.

**Table H3-4. Summary of exceedance probabilities for piscivorus birds from Calcasieu Estuary.**

| Location | Probability of Exceedance (%) | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Average-Sized Piscivorus Birds | | | | | | Small Piscivorus Birds | | | | | |
| | *LB* | | *FOMC* | | *UB* | | *LB* | | *FOMC* | | *UB* | |
| | LT | UT | LT | UT | LT | UT | LT | UT | LT | UT | LT | UT |
| ***TCDD – TEQs*** | | | | | | | | | | | | |
| Bayou d'Inde | 24 | 0 | 67 | 0 | 100 | 1 | 89 | 0 | 99 | 0 | 100 | 1 |
| Middle Calcasieu River | 0 | 0 | 4 | 0 | 100 | 1 | 24 | 0 | 59 | 0 | 100 | 4 |
| Reference Areas | 0 | 0 | 0 | 0 | 100 | 1 | 22 | 0 | 34 | 0 | 100 | 4 |
| ***Selenium*** | | | | | | | | | | | | |
| Bayou d'Inde | 0 | 0 | 7 | 0 | 64 | 7 | 36 | 0 | 72 | 2 | 100 | 55 |
| Reference Areas | 0 | 0 | 6 | 0 | 75 | 12 | 20 | 0 | 68 | 2 | 100 | 67 |
| ***Mercury*** | | | | | | | | | | | | |
| Bayou d'Inde | 0 | 0 | 9 | 0 | 70 | 1 | 34 | 0 | 77 | 0 | 100 | 2 |
| Upper Calcasieu River | 0 | 0 | 0 | 0 | 5 | 0 | 0 | 0 | 0 | 0 | 42 | 1 |
| Middle Calcasieu River | 0 | 0 | 0 | 0 | 7 | 0 | 0 | 0 | 0 | 0 | 53 | 1 |
| Reference Areas | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 8 | 0 |
| ***Total PCBs*** | | | | | | | | | | | | |
| Bayou d'Inde | 0 | 0 | 0 | 0 | 32 | 0 | 0 | 0 | 20 | 0 | 96 | 0 |
| Reference Areas | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 3 | 0 |

LB = Lower probability Bound; FOMC = First Order Monte Carlo; UB = Upper Probability Bound; LT = Lower Toxicity Threshold; UT = Upper Toxicity Threshold; TCDD = tetrachlorodibenzo-*p* -dioxin; TEQs = toxic equivalents; PCBs = polychlorinated biphenyls.

**Table H3-5. Summary of exceedance probabilities for the Appendix G benchmarks.**

| Location | | Probability of Exceedance (%) | | | | | |
|---|---|---|---|---|---|---|---|
| | | Average-Sized Piscivorus Bird | | | Small Piscivorus Birds | | |
| | *Benchmark* | *LB* | *FOMC* | *UB* | *LB* | *FOMC* | *UB* |
| ***TCDD – TEQs*** | | | | | | | |
| Bayou d'Inde | 44.3 ng/kg bw/d | 0 | 0 | 57 | 3 | 37 | 100 |
| Middle Calcasieu River | | 0 | 0 | 43 | 0 | 0 | 87 |
| Reference Areas | | 0 | 0 | 42 | 0 | 0 | 85 |
| ***Selenium*** | | | | | | | |
| Bayou d'Inde | 0.707 mg/kg bw/d | 0 | 0 | 3 | 0 | 0 | 33 |
| Reference Areas | | 0 | 0 | 6 | 0 | 0 | 44 |
| ***Mercury*** | | | | | | | |
| Bayou d'Inde | 0.0202 mg/kg bw/d | 61 | 91 | 100 | 98 | 100 | 100 |
| Upper Calcasieu River | | 0 | 6 | 69 | 25 | 69 | 100 |
| Middle Calcasieu River | | 0 | 9 | 78 | 37 | 78 | 100 |
| Reference Areas | | 0 | 0 | 23 | 0 | 9 | 88 |
| ***Total PCBs*** | | | | | | | |
| Bayou d'Inde | 0.569 mg/kg bw/d | 0 | 0 | 1 | 0 | 0 | 3 |
| Reference Areas | | 0 | 0 | 0 | 0 | 0 | 0 |

LB = Lower probability Bound; FOMC = First Order Monte Carlo; UB = Upper Probability Bound; LT = Lower Toxicity Threshold; UT = Upper Toxicity Threshold.

# Figures

**Figure H3-1. Overview of approach used to assess exposure of piscivorus birds to contaminants of concern (COCs) in the Calcasieu Estuary.**

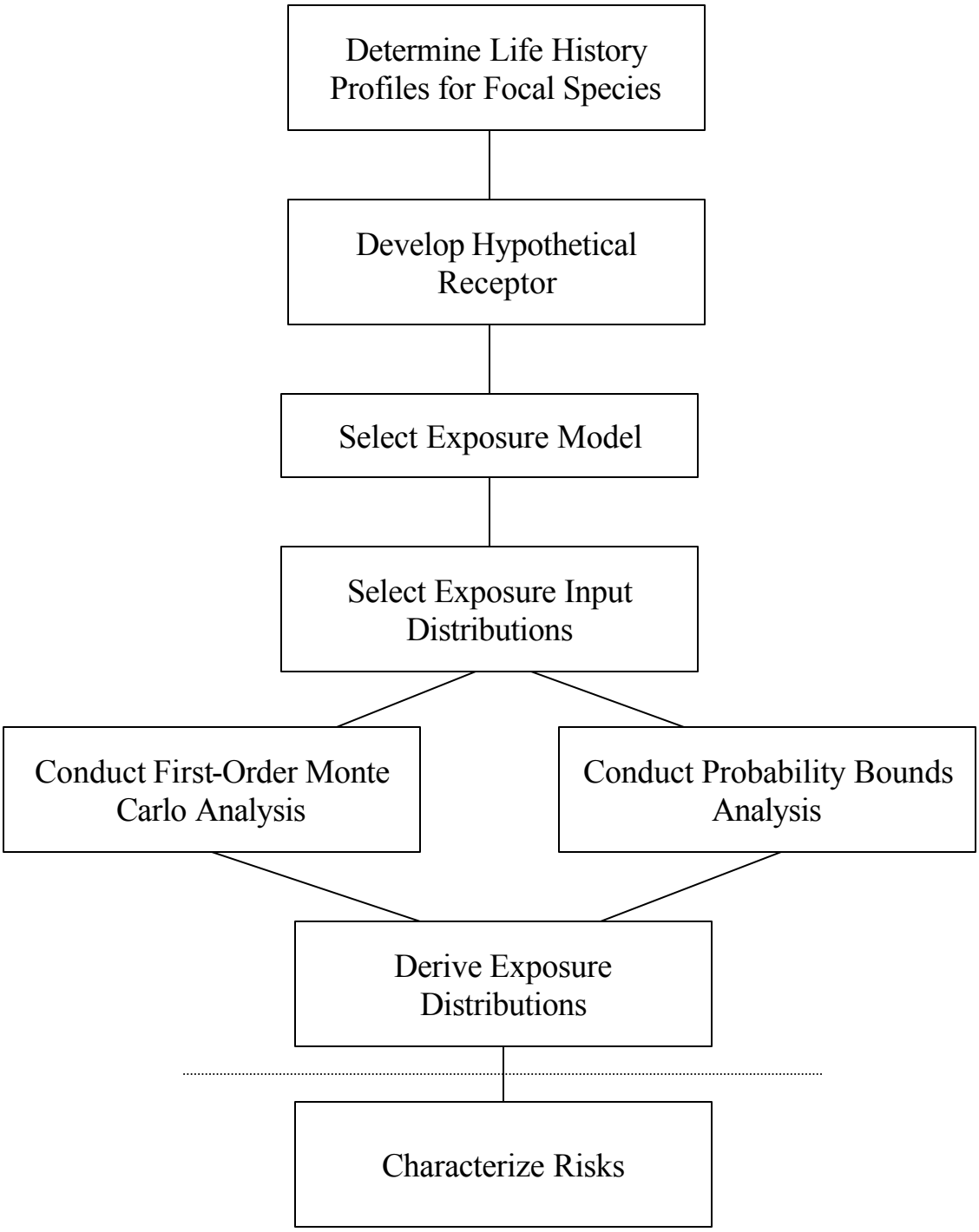

**Figure H3-2. Overview of approach used to assess the effects to piscivorus birds exposed to contaminants of concern (COCs) in the Calcasieu Estuary.**

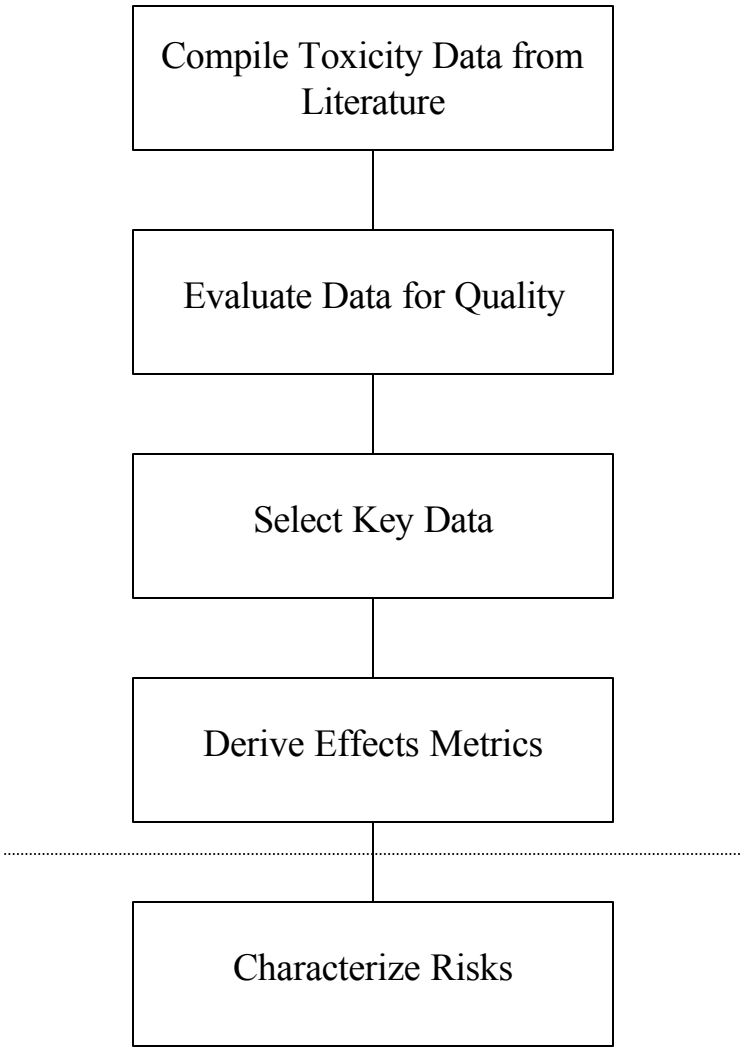

**Figure H3-3. Overview of approach used to assess the risks to piscivorus birds exposed to contaminants of concern (COCs) in the Calcasieu Estuary.**

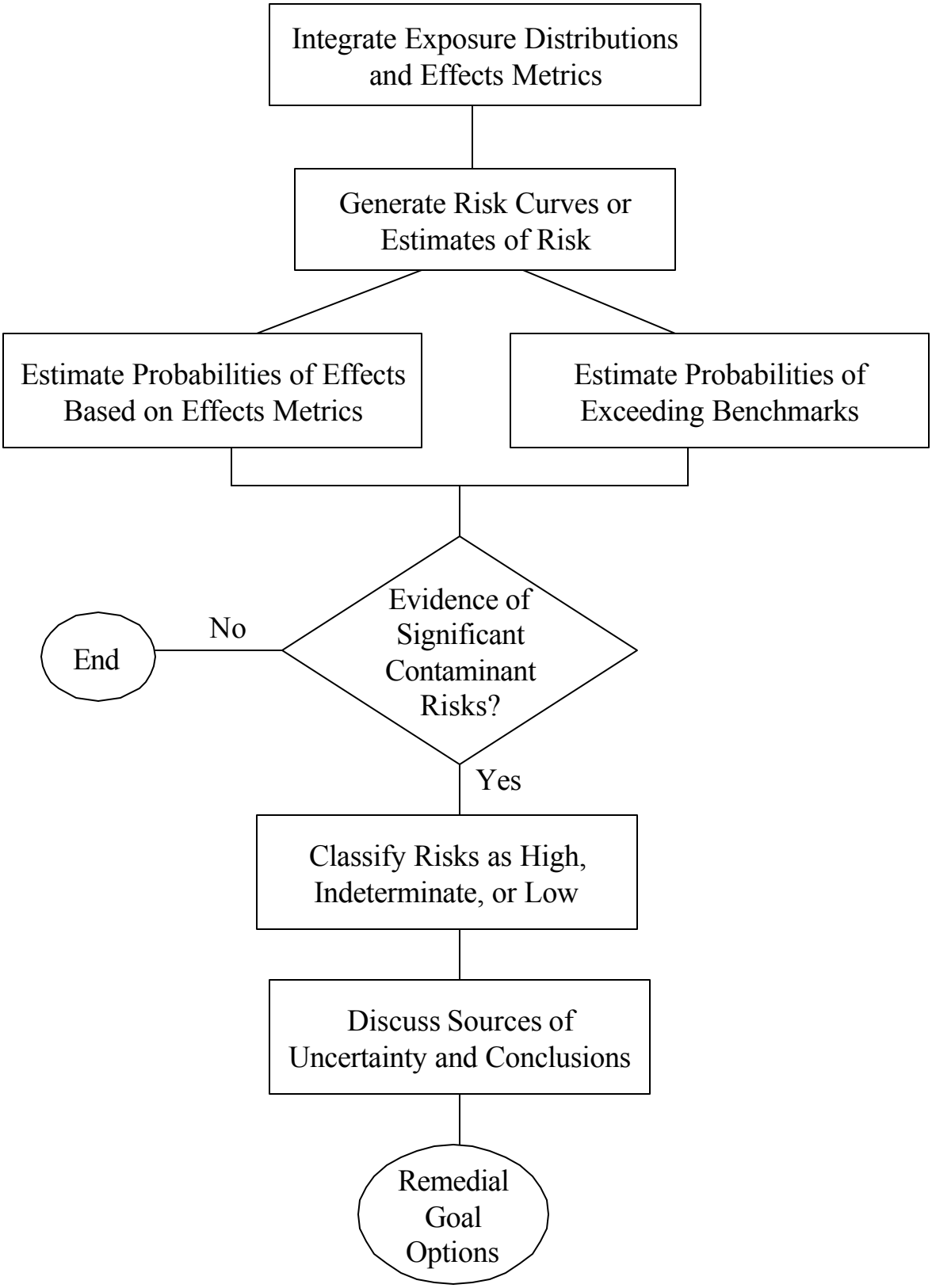

**Figure H3-4. Reverse cumulative probability distribution of total daily intake rates of TCDD-TEQs by average-sized piscivorus birds in Bayou d'Inde, Calcasieu Estuary.**

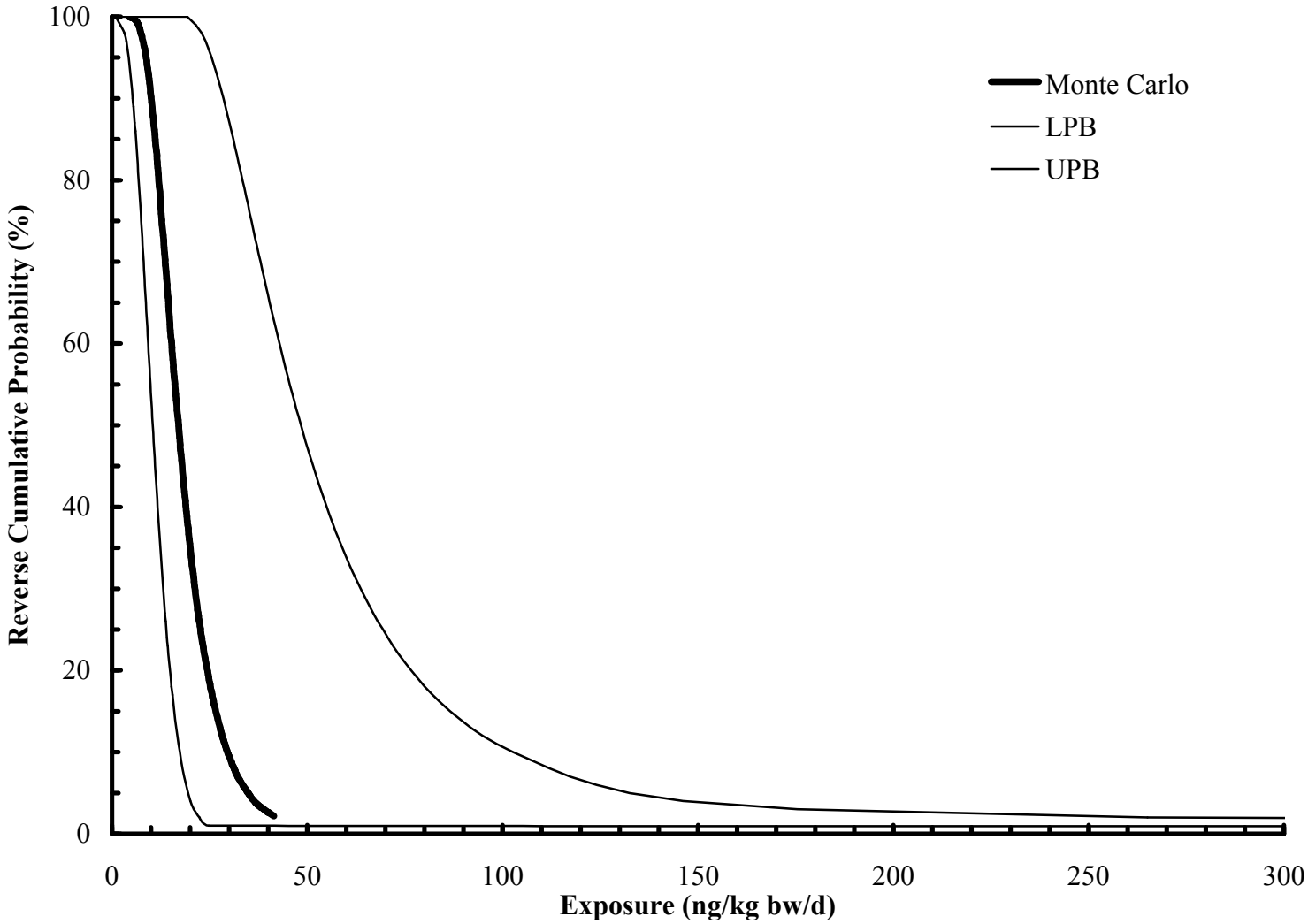

**Figure H3-5. Reverse cumulative probability distribution of total daily intake rates of TCDD-TEQs by small piscivorus birds in Bayou d'Inde, Calcasieu Estuary.**

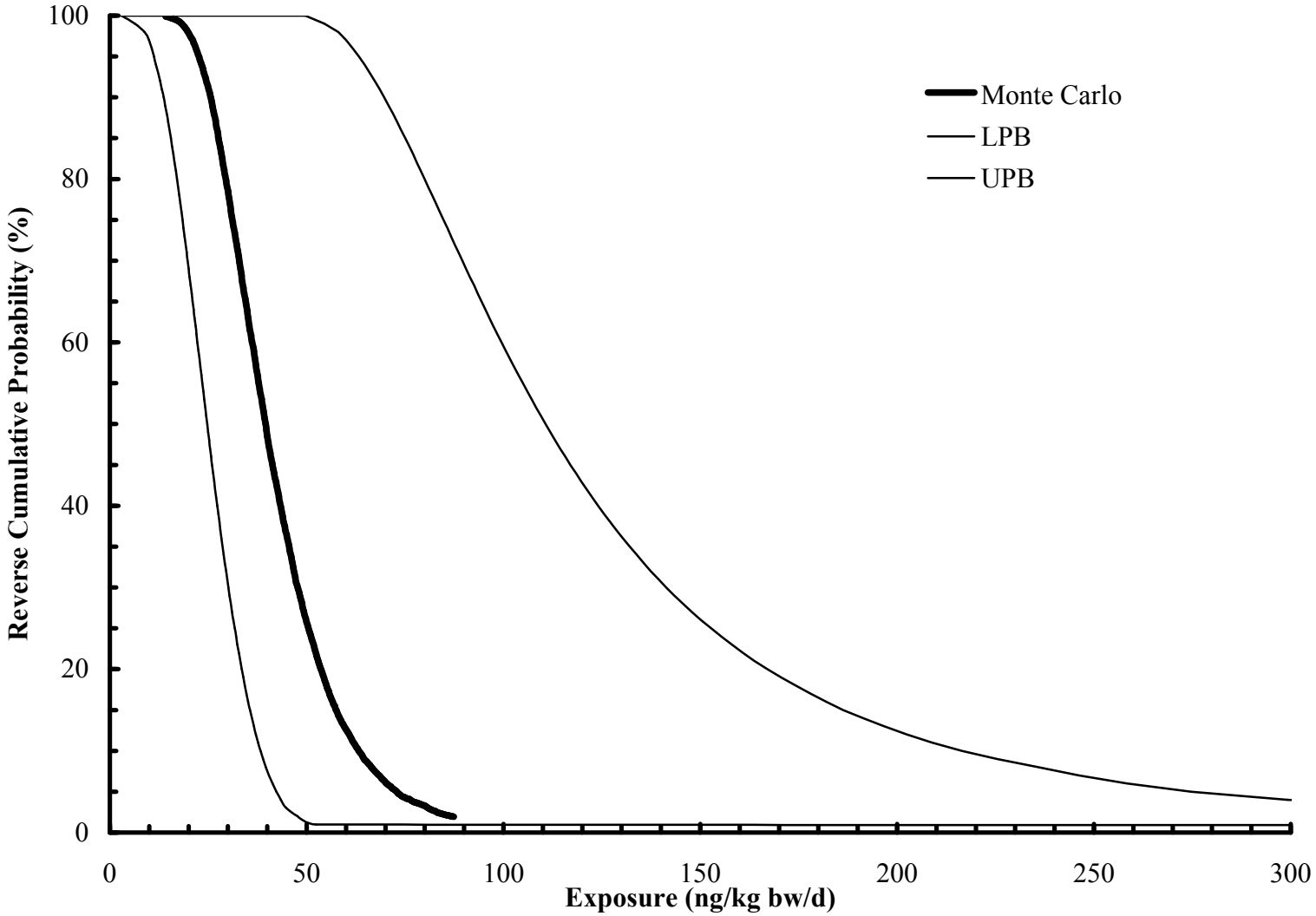

**Figure H3-6. Reverse cumulative probability distribution of total daily intake rates of TCDD-TEQs by average-sized piscivorus birds in the Middle Calcasieu River, Calcasieu Estuary.**

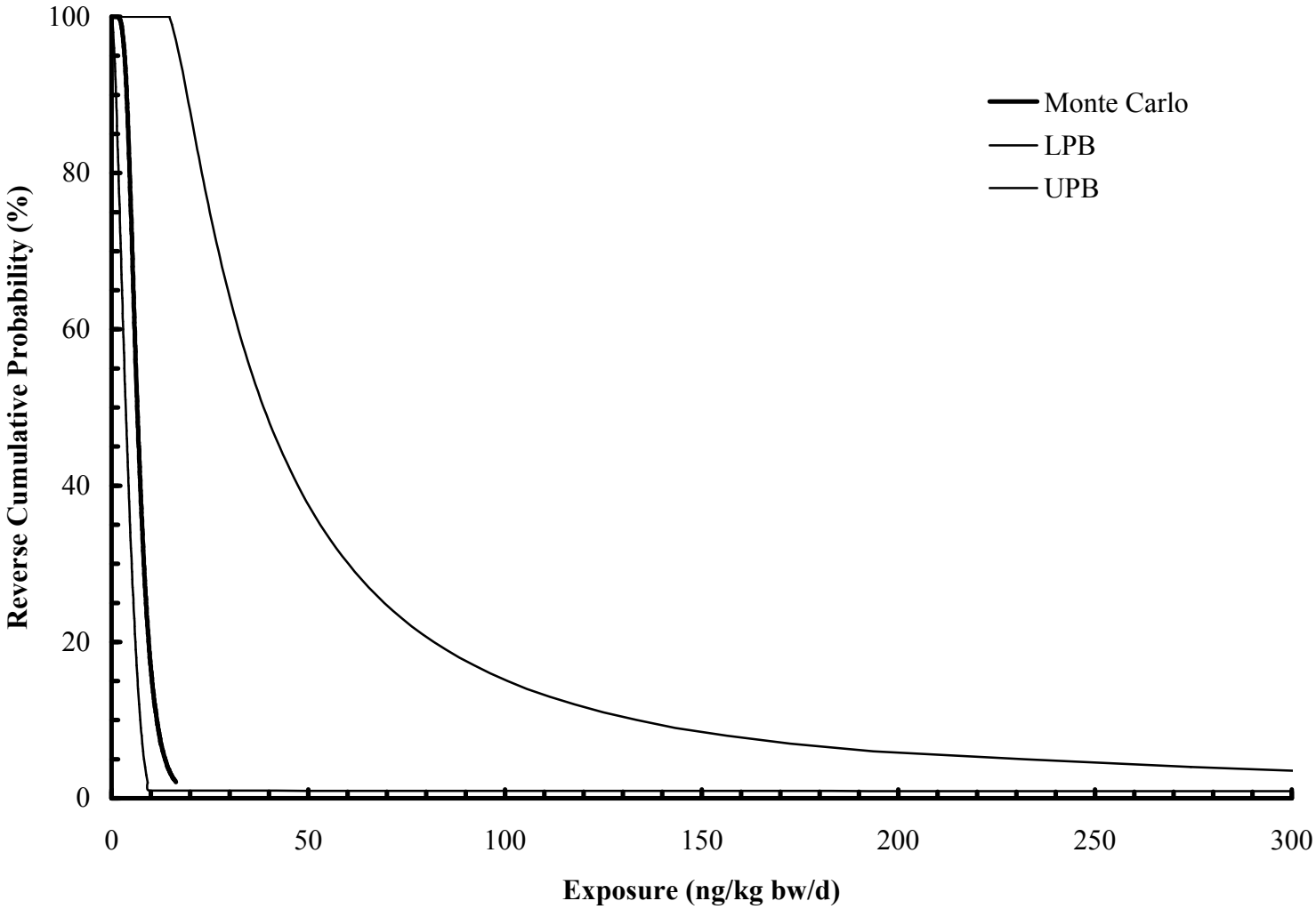

**Figure H3-7. Reverse cumulative probability distribution of total daily intake rates of TCDD-TEQs by small piscivorus birds in the Middle Calcasieu River, Calcasieu Estuary.**

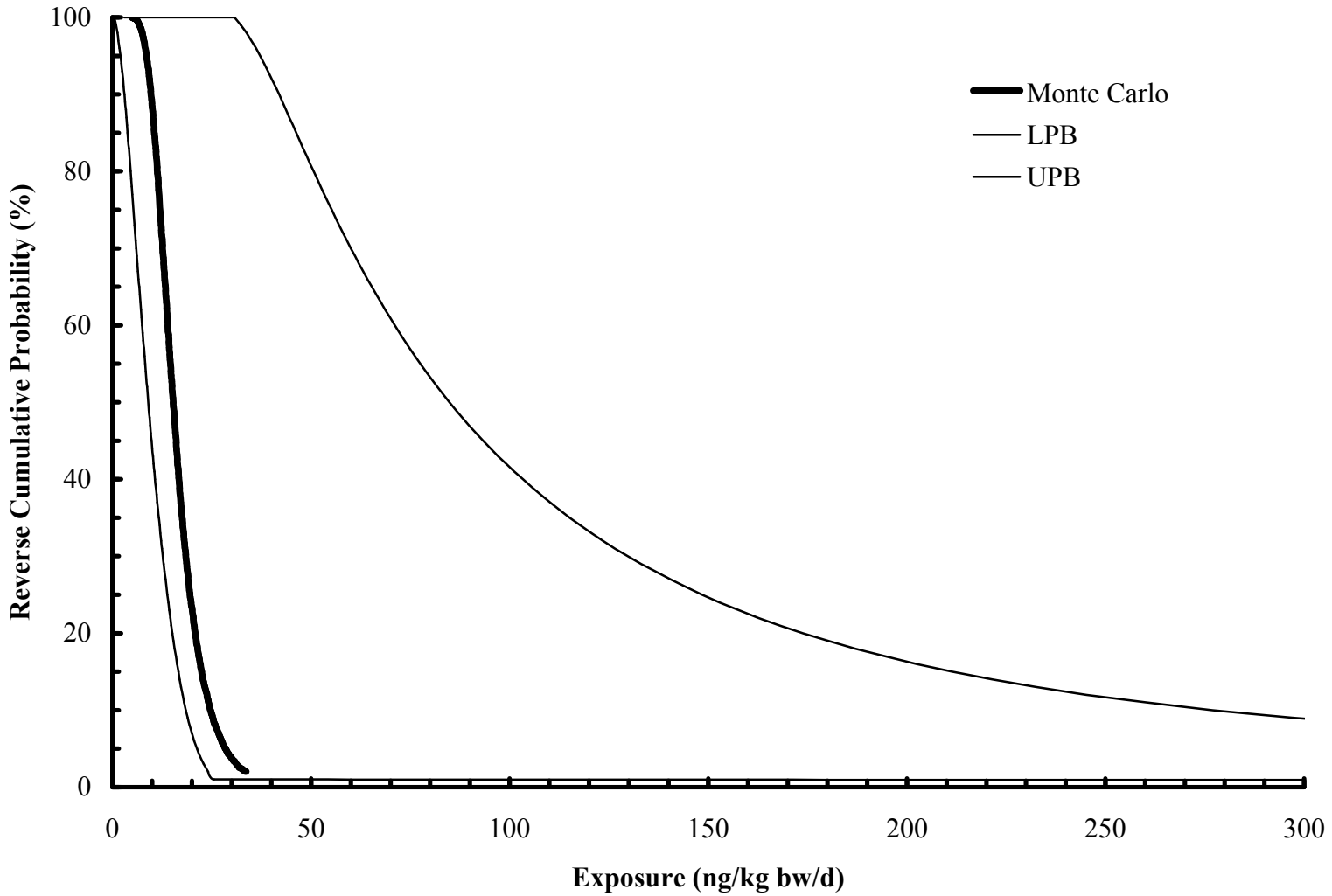

**Figure H3-8. Reverse cumulative probability distribution of total daily intake rates of TCDD-TEQs by average-sized piscivorus birds in the reference areas, Calcasieu Estuary.**

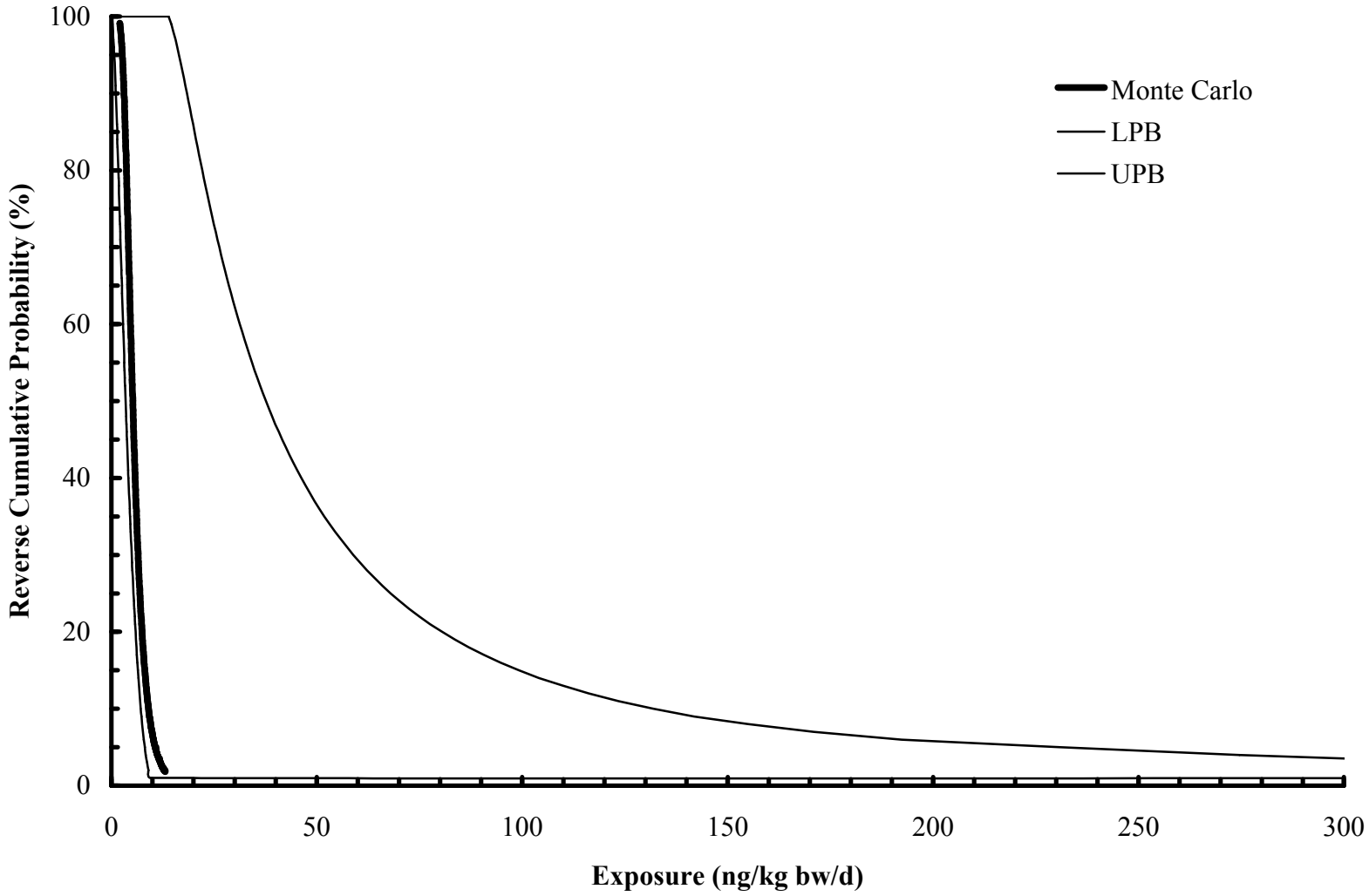

**Figure H3-9. Reverse cumulative probability distribution of total daily intake rates of TCDD-TEQs by small piscivorus birds in the reference areas, Calcasieu Estuary.**

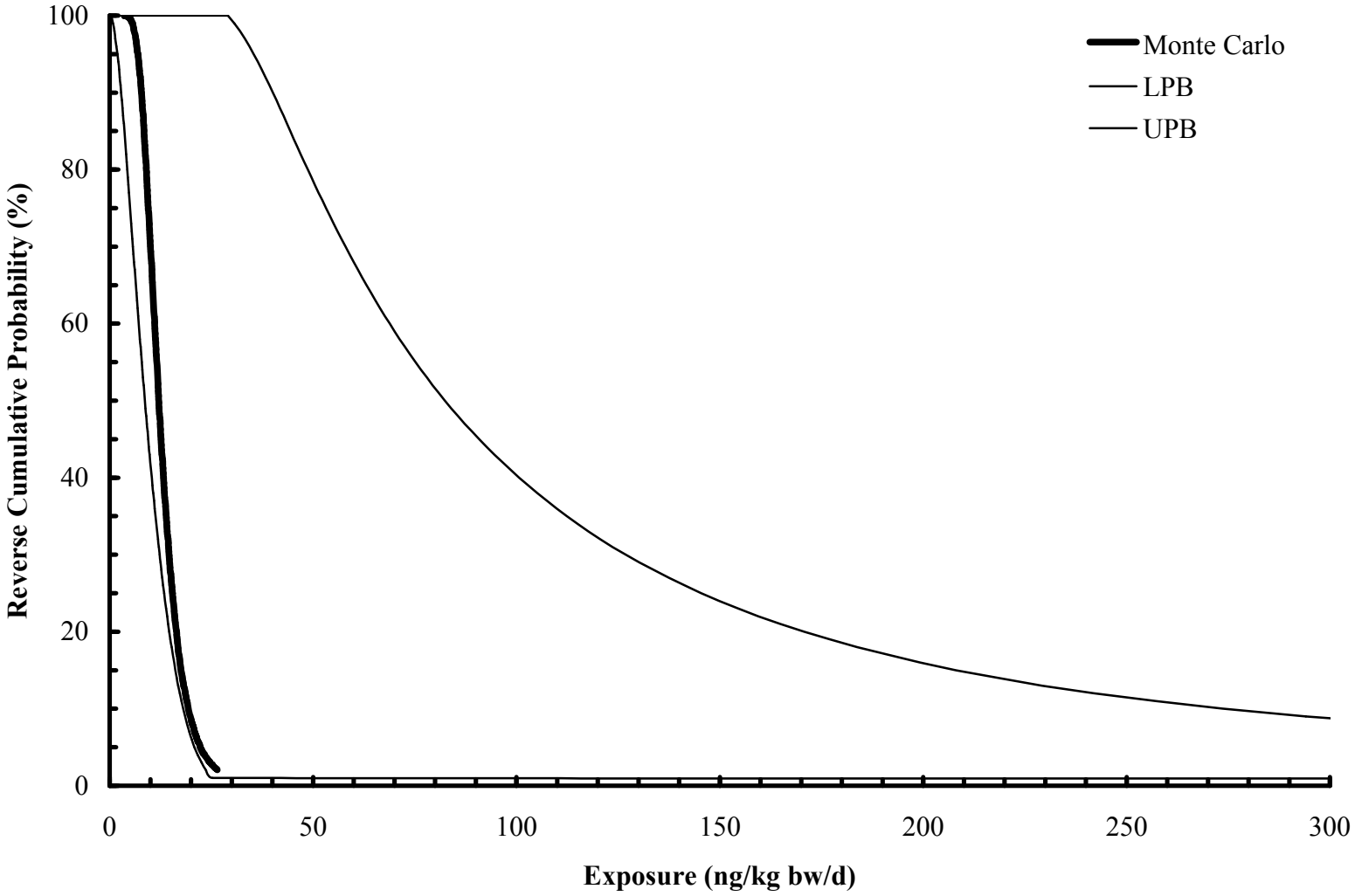

**Figure H3-10. Reverse cumulative probability distribution of total daily intake rates of selenium by average-sized piscivorus birds in Bayou d'Inde, Calcasieu Estuary.**

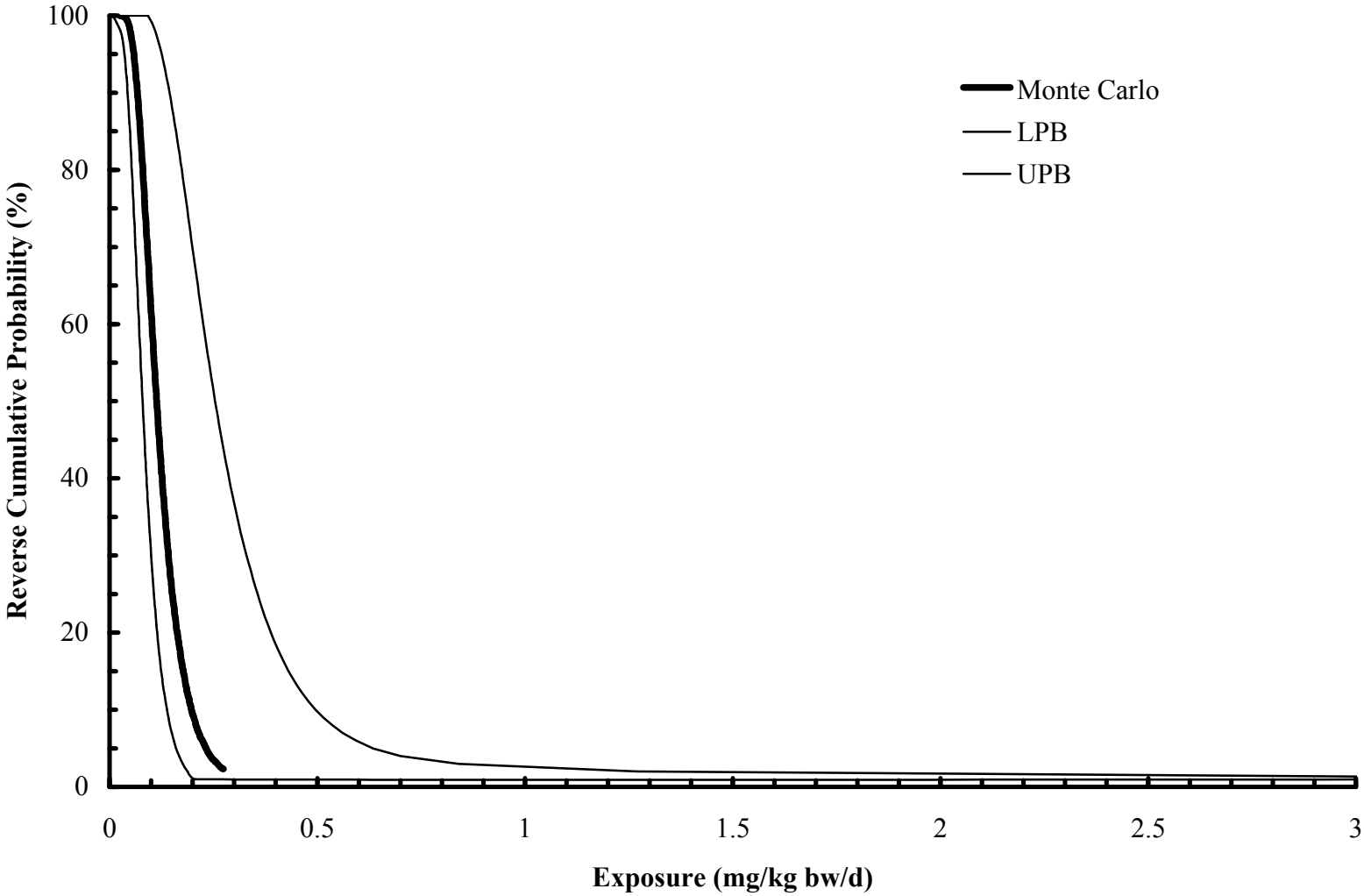

**Figure H3-11. Reverse cumulative probability distribution of total daily intake rates of selenium by small piscivorus birds in Bayou d'Inde, Calcasieu Estuary.**

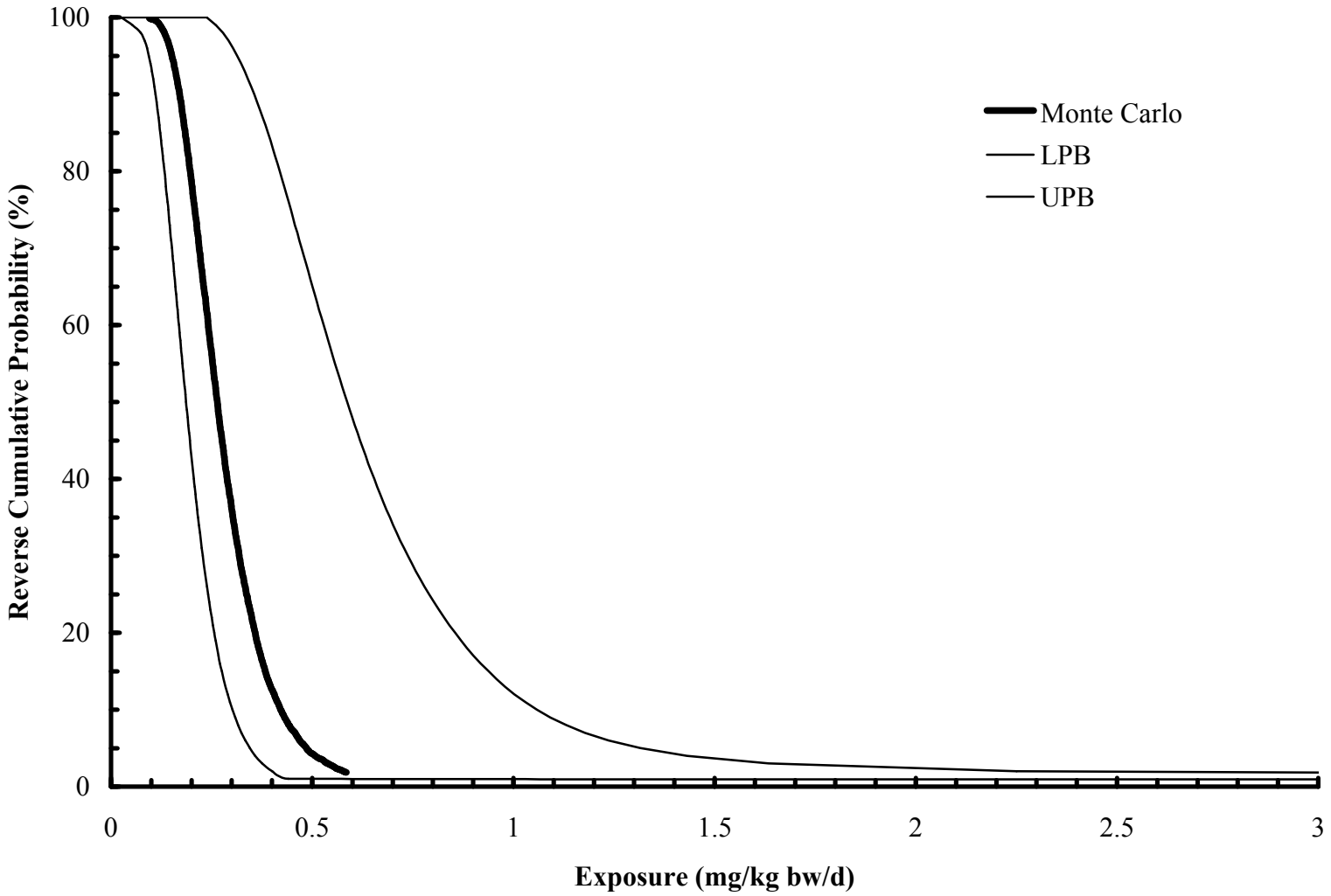

**Figure H3-12. Reverse cumulative probability distribution of total daily intake rates of selenium by average-sized piscivorus birds in the reference areas, Calcasieu Estuary.**

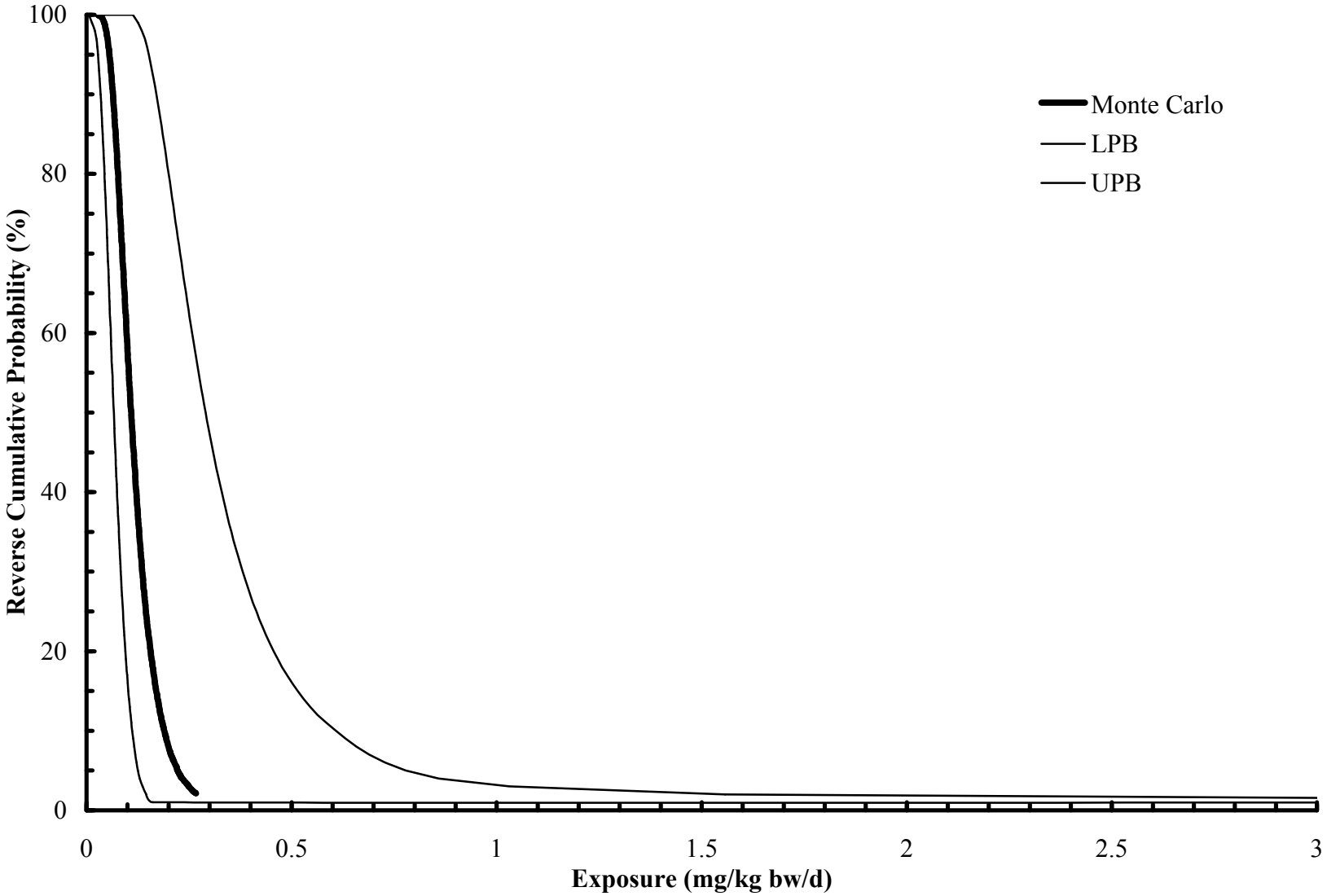

**Figure H3-13. Reverse cumulative probability distribution of total daily intake rates of selenium by small piscivorus birds in the reference areas, Calcasieu Estuary.**

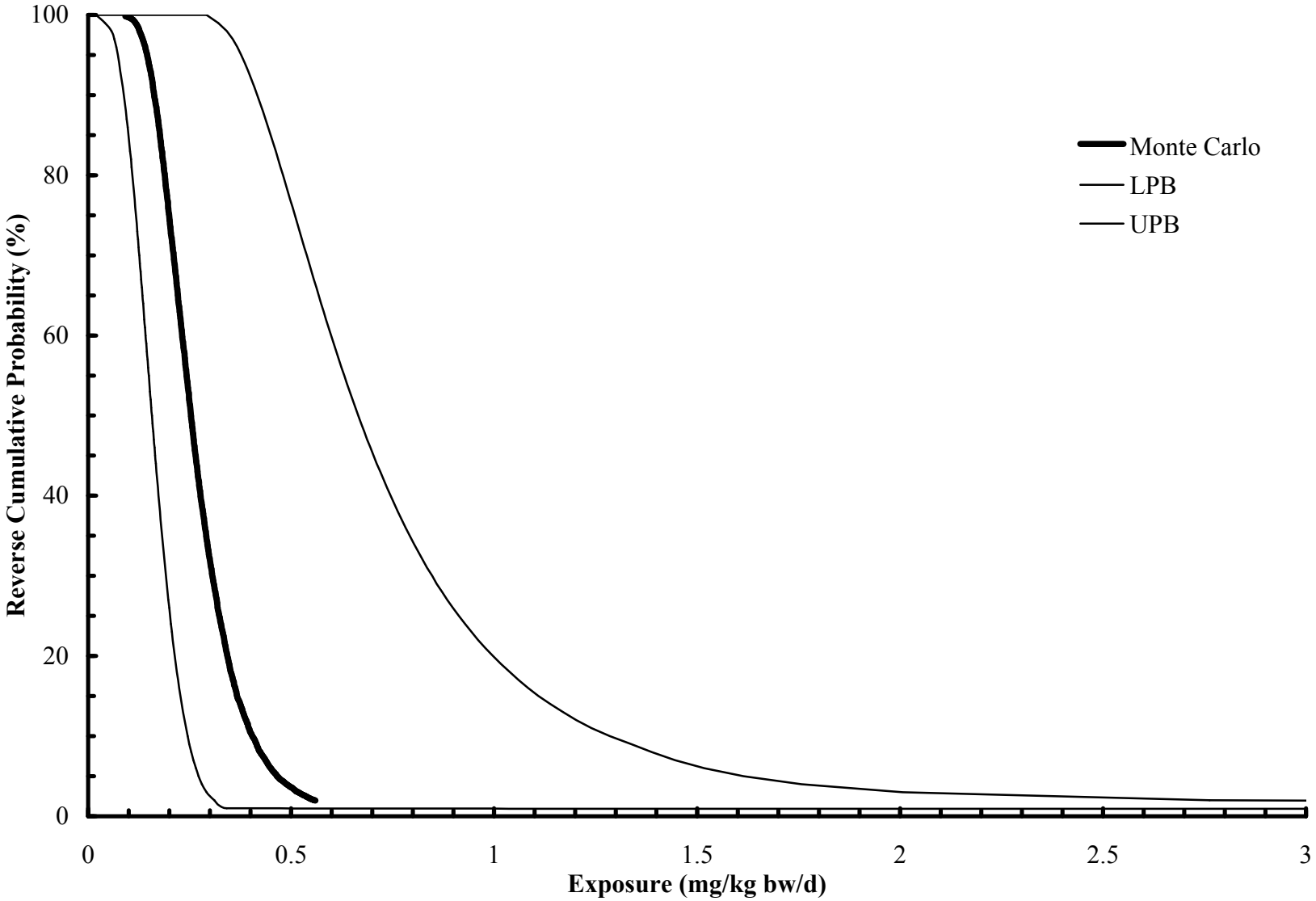

**Figure H3-14. Reverse cumulative probability distribution of total daily intake rates of mercury by average-sized piscivorus birds in Bayou d'Inde, Calcasieu Estuary.**

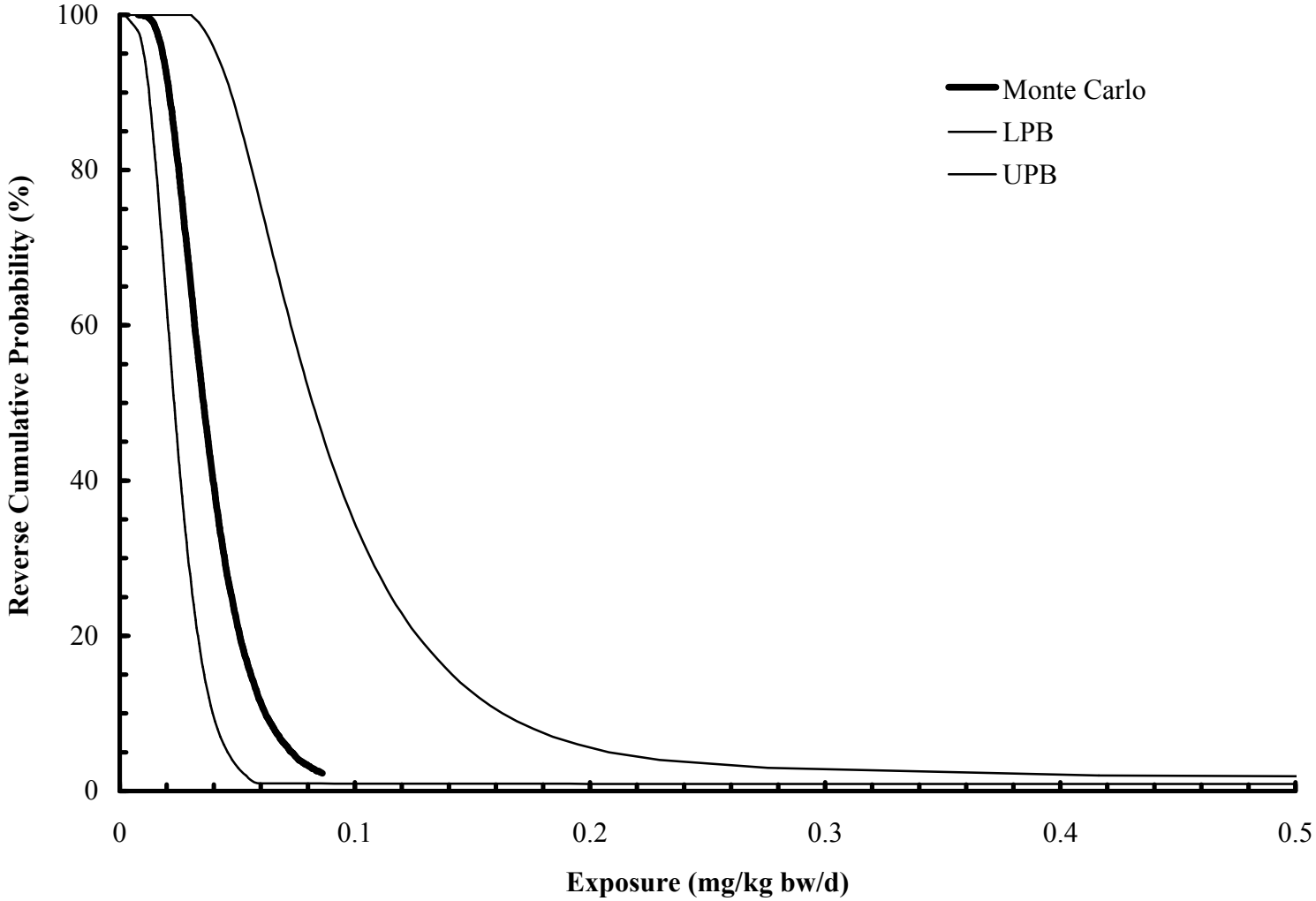

**Figure H3-15. Reverse cumulative probability distribution of total daily intake rates of mercury by small piscivorus birds in Bayou d'Inde, Calcasieu Estuary.**

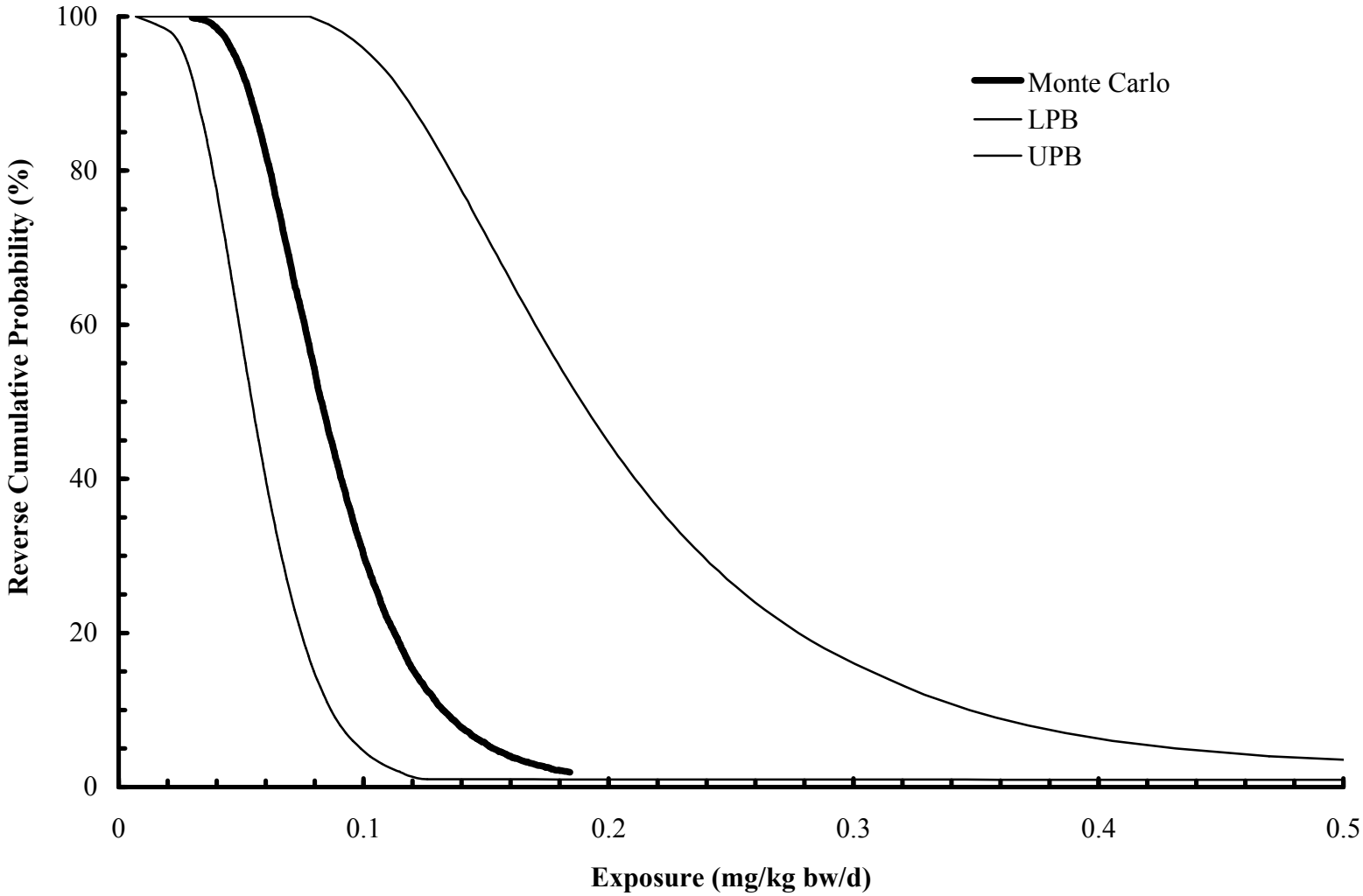

**Figure H3-16. Reverse cumulative probability distribution of total daily intake rates of mercury by average-sized piscivorus birds in the Upper Calcasieu River, Calcasieu Estuary.**

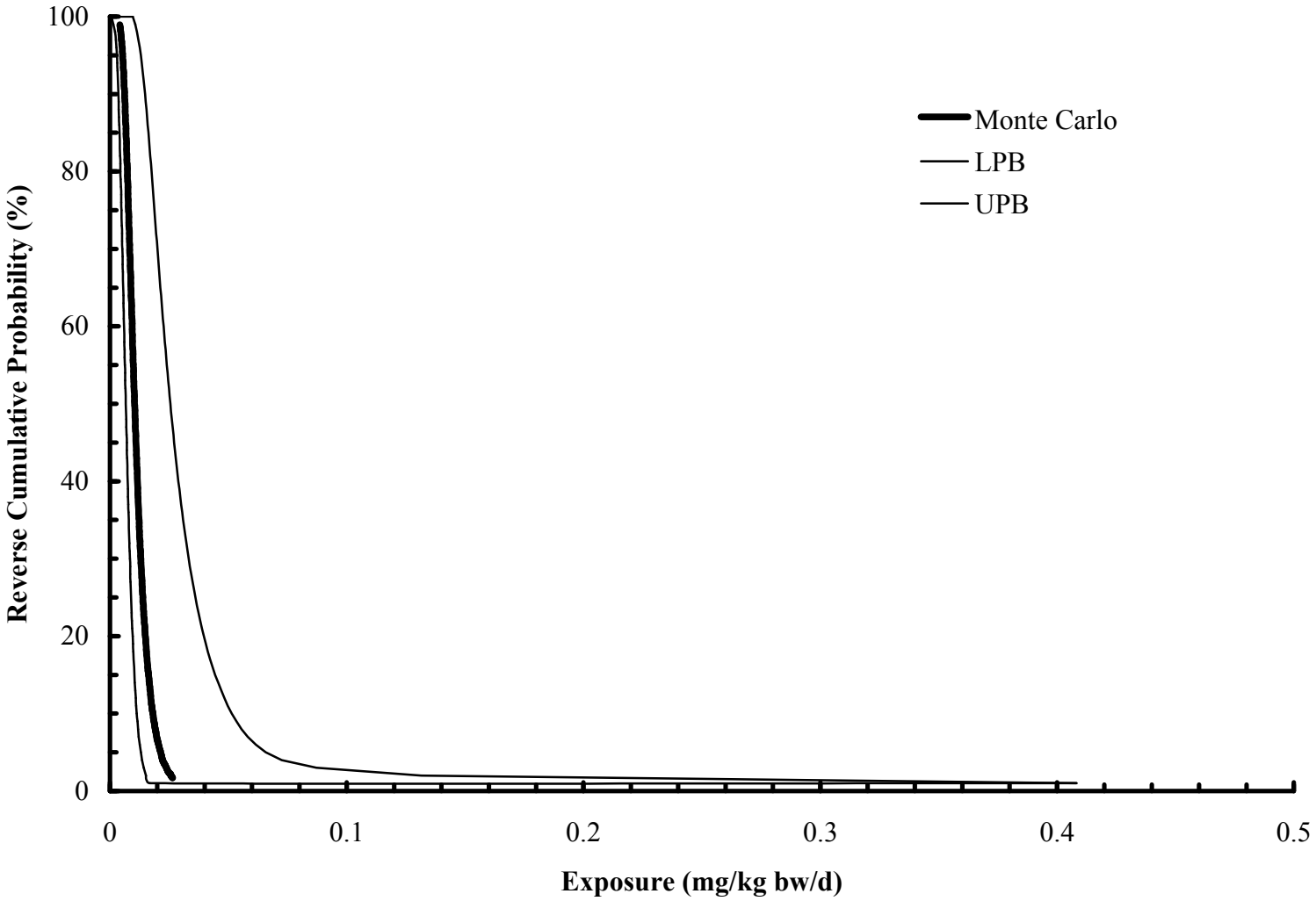

**Figure H3-17. Reverse cumulative probability distribution of total daily intake rates of mercury by small piscivorus birds in the Upper Calcasieu River, Calcasieu Estuary.**

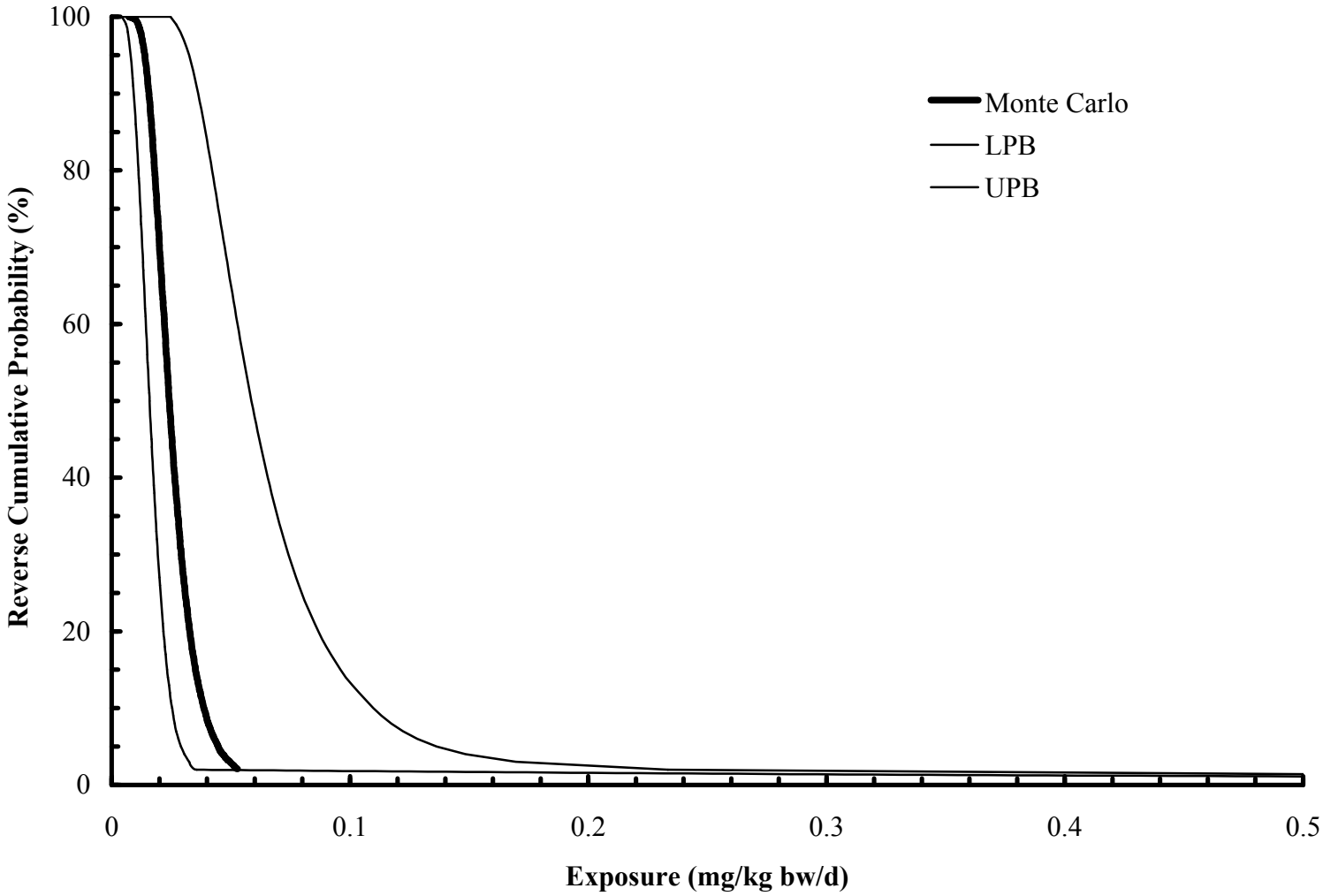

**Figure H3-18. Reverse cumulative probability distribution of total daily intake rates of mercury by average-sized piscivorus birds in the Middle Calcasieu River, Calcasieu Estuary.**

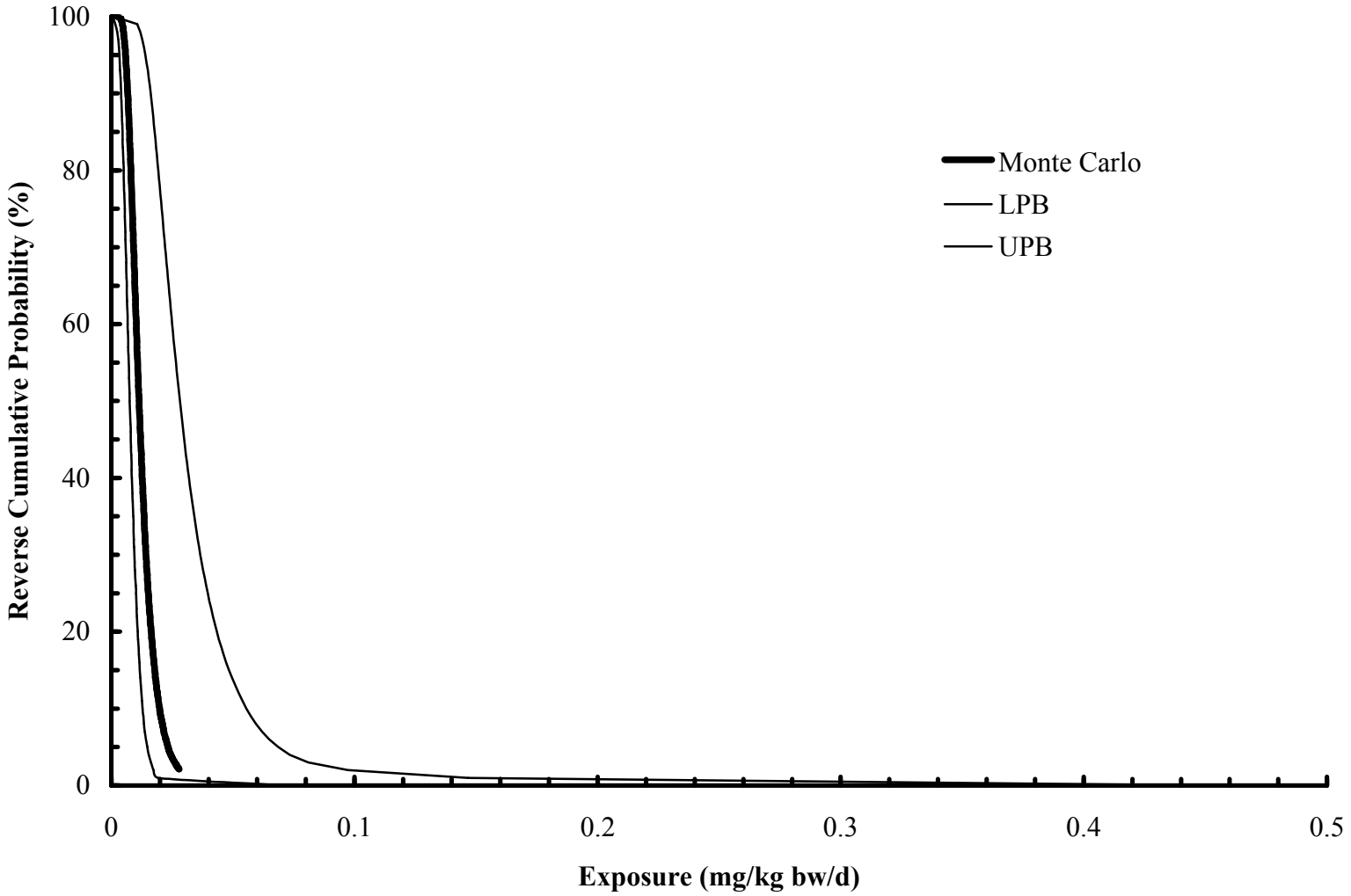

**Figure H3-19. Reverse cumulative probability distribution of total daily intake rates of mercury by small piscivorus birds in the Middle Calcasieu River, Calcasieu Estuary.**

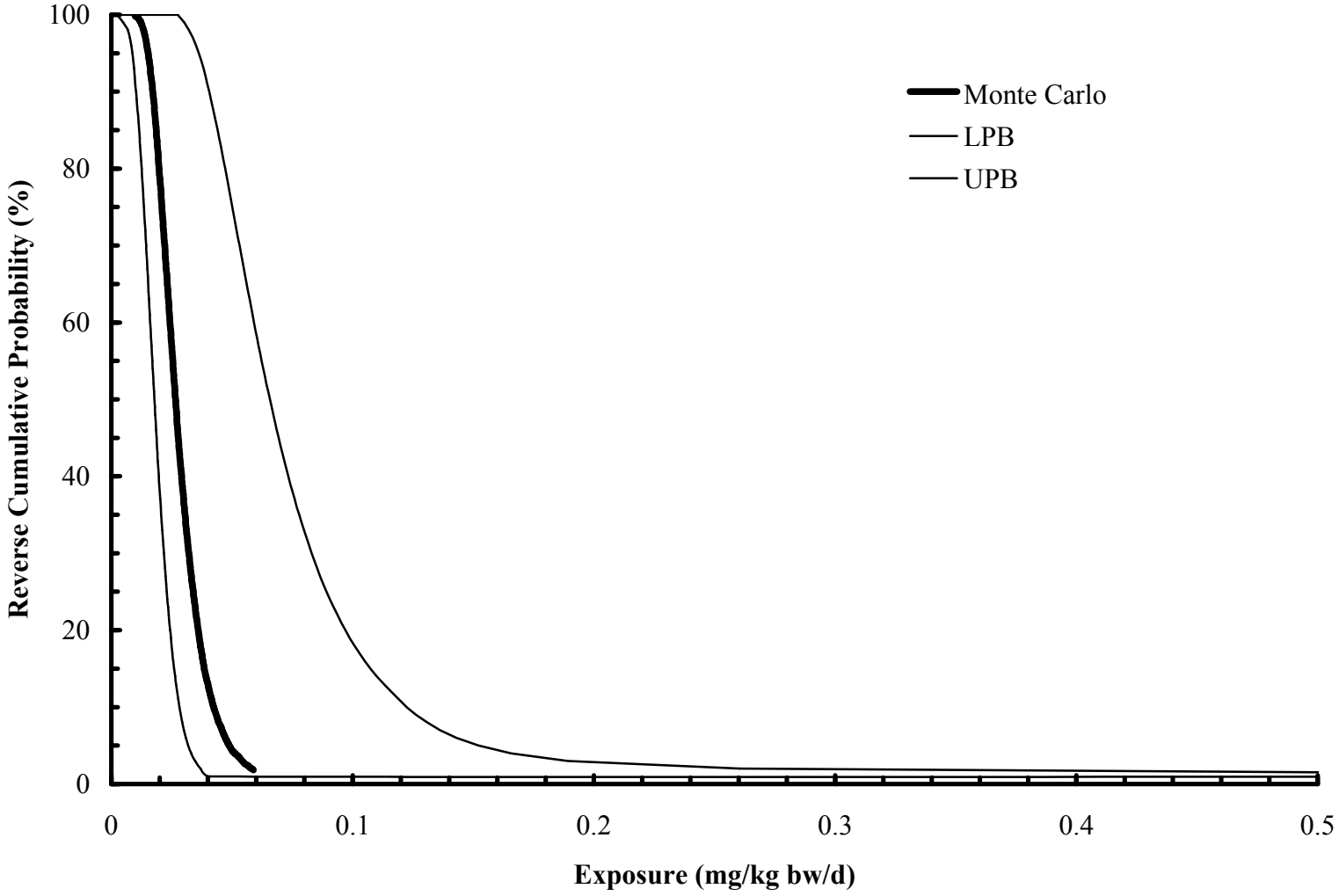

**Figure H3-20. Reverse cumulative probability distribution of total daily intake rates of mercury by average-sized piscivorus birds in the reference areas, Calcasieu Estuary.**

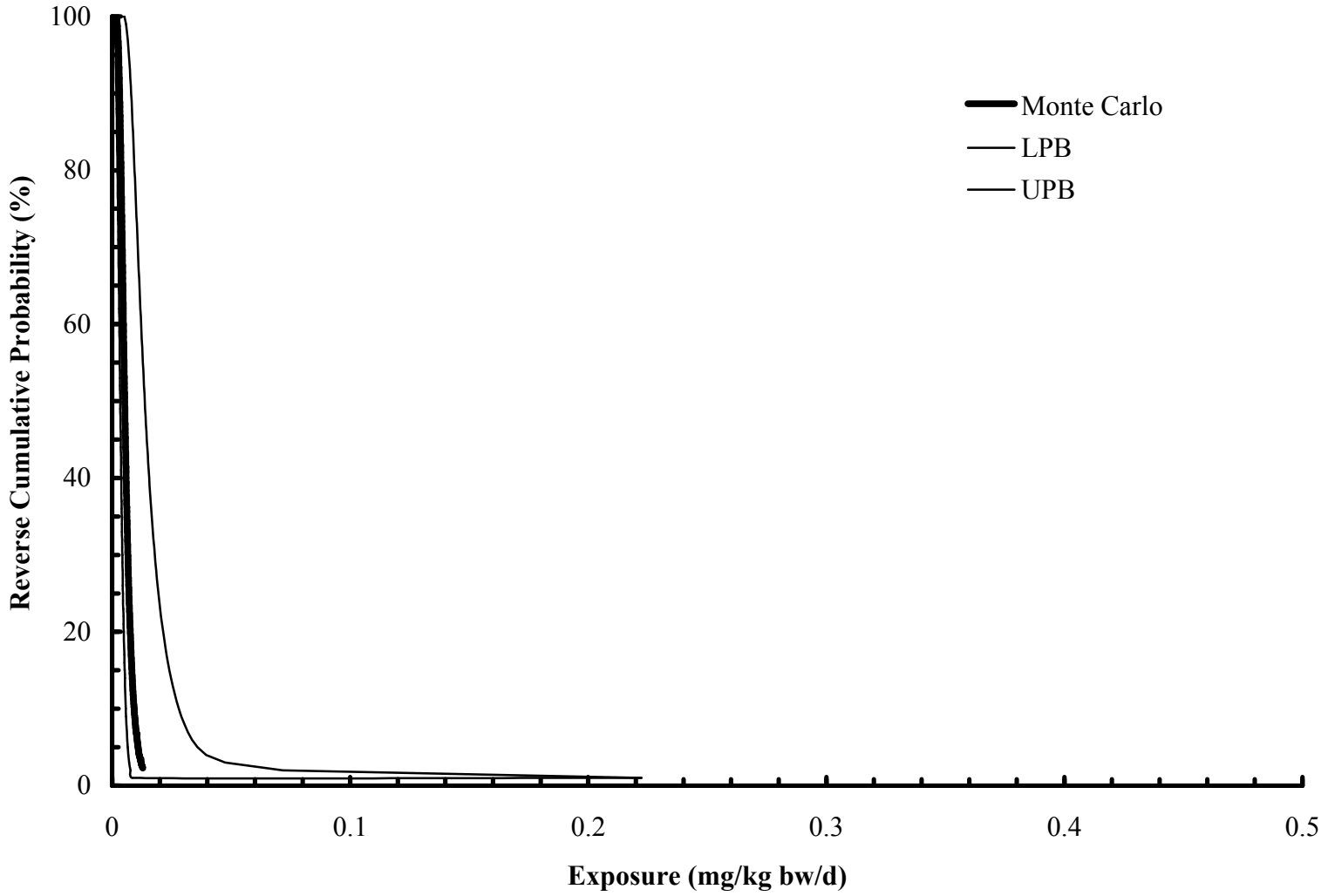

**Figure H3-21. Reverse cumulative probability distribution of total daily intake rates of mercury by small piscivorus birds in the reference areas, Calcasieu Estuary.**

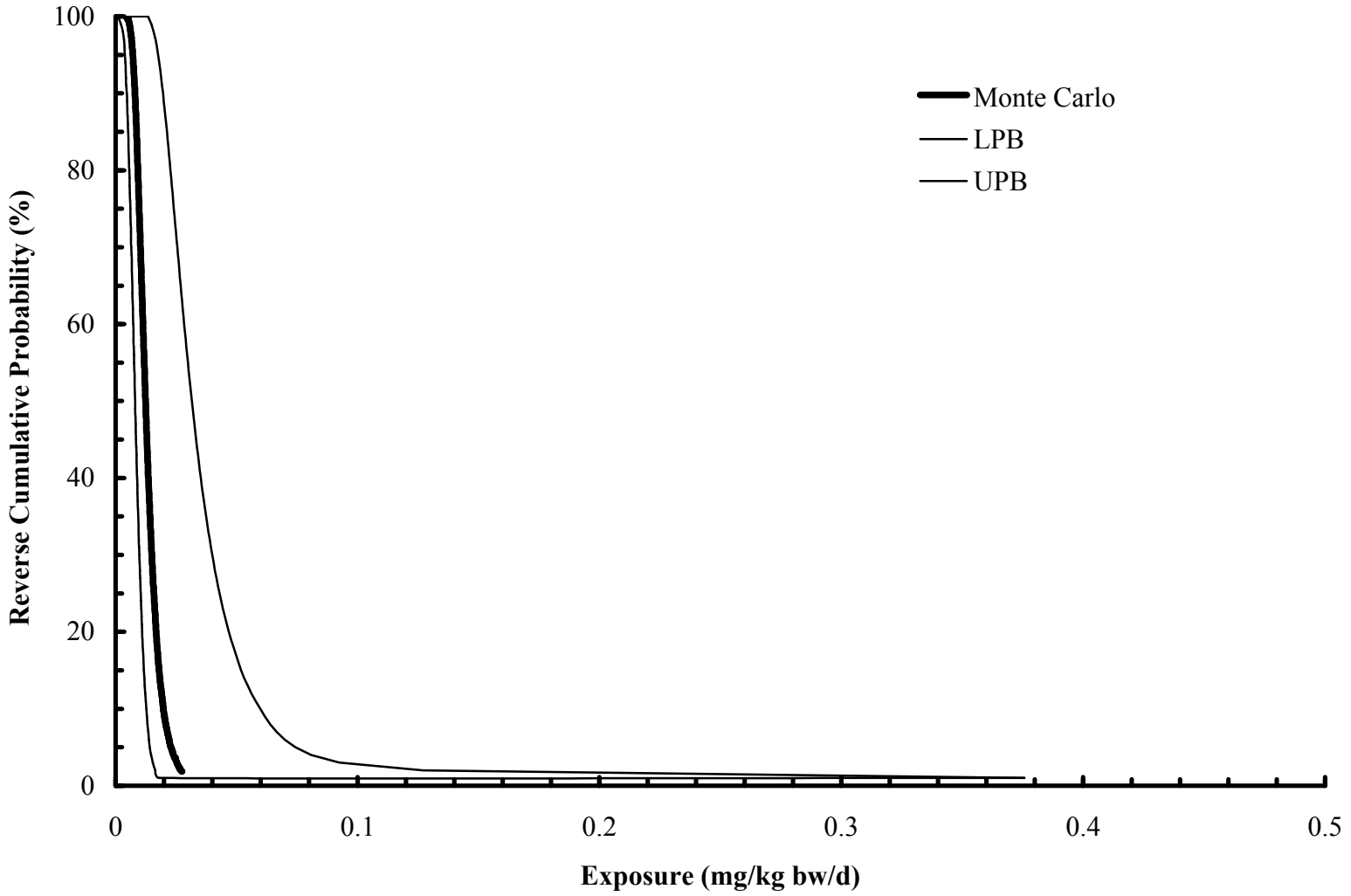

**Figure H3-22. Reverse cumulative probability distribution of total daily intake rates of total PCBs by average-sized piscivorus birds in Bayou d'Inde, Calcasieu Estuary.**

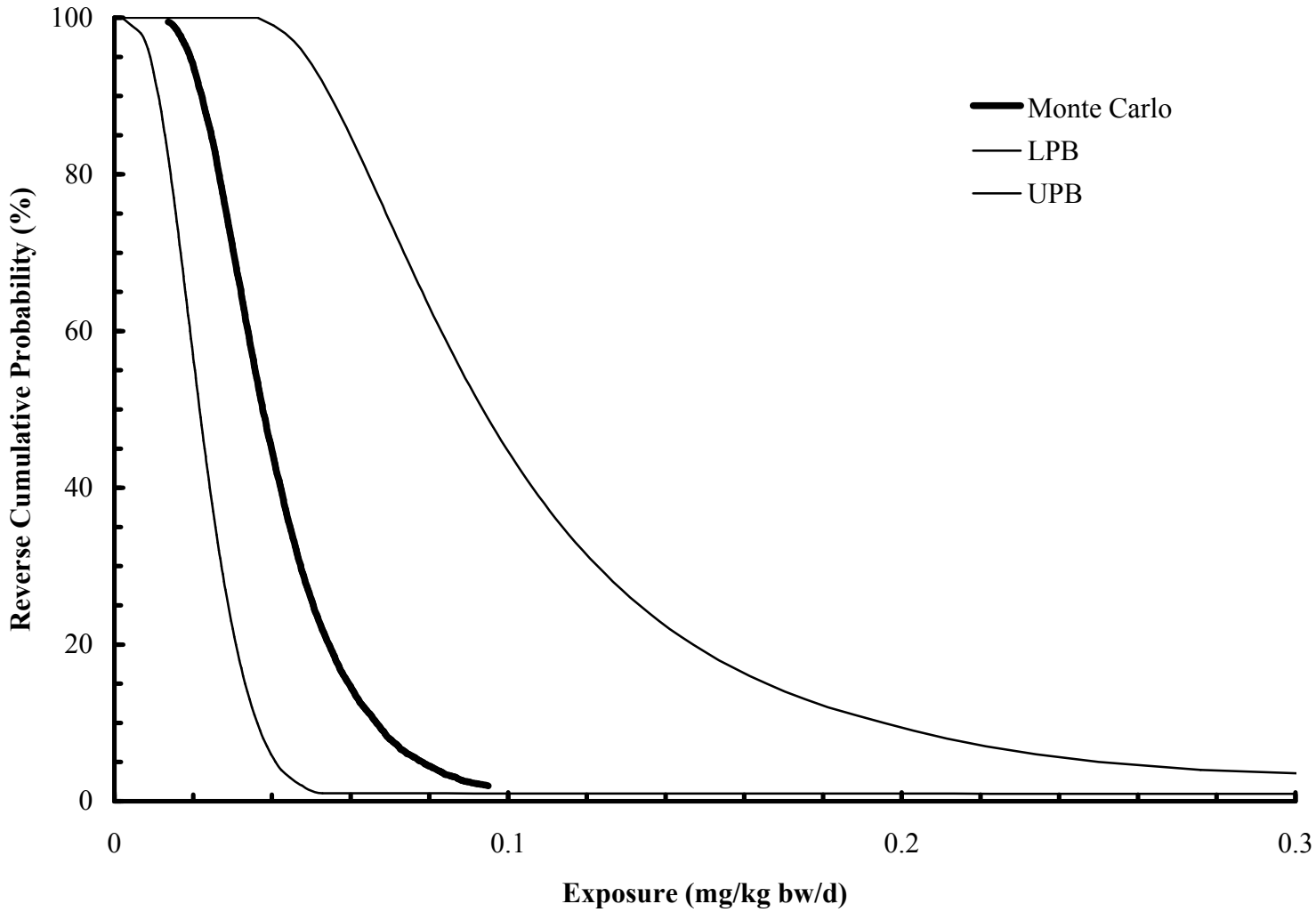

**Figure H3-23. Reverse cumulative probability distribution of total daily intake rates of total PCBs by small piscivorus birds in Bayou d'Inde, Calcasieu Estuary.**

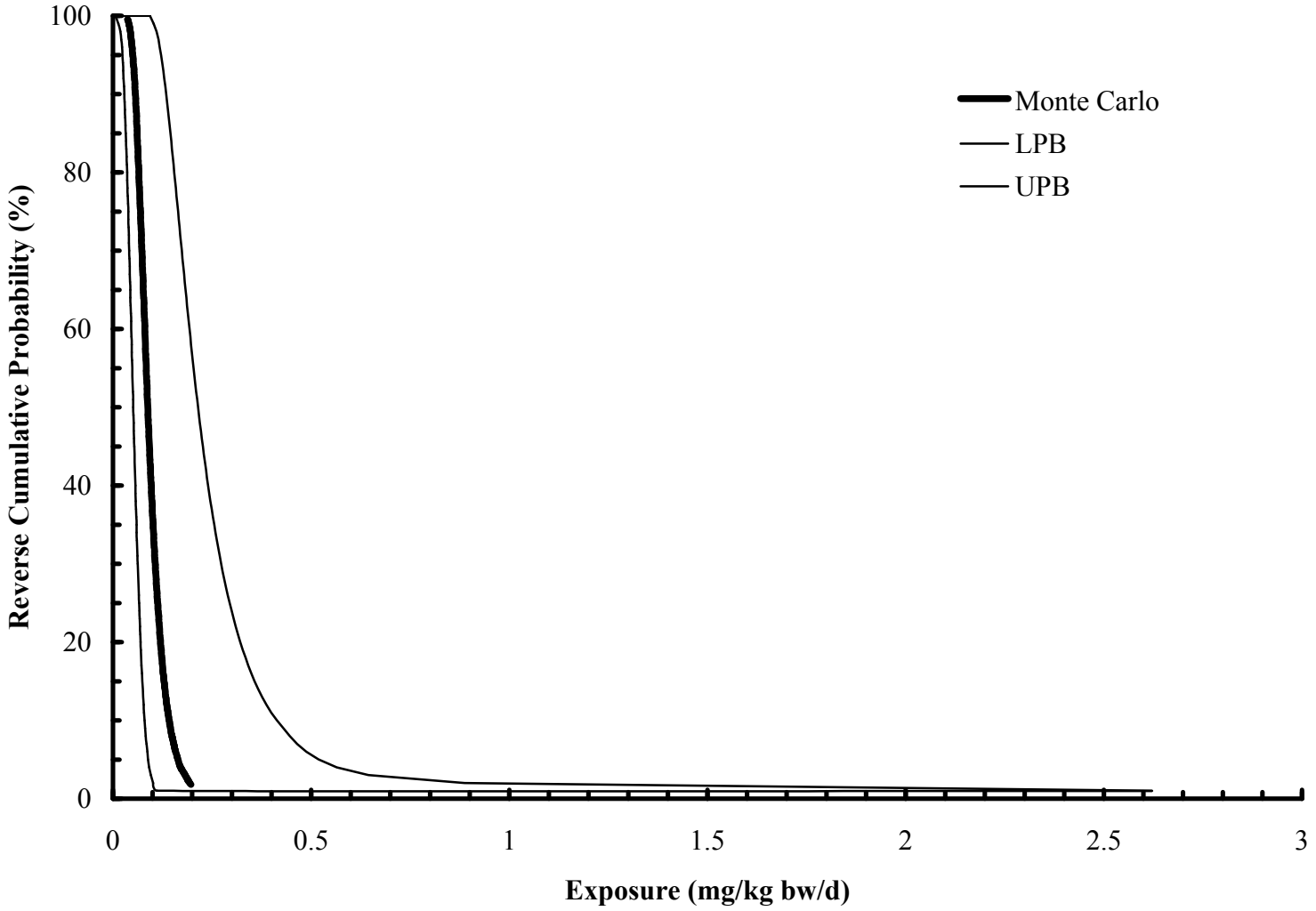

**Figure H3-24. Reverse cumulative probability distribution of total daily intake rates of total PCBs by average-sized piscivorus birds in the reference areas, Calcasieu Estuary.**

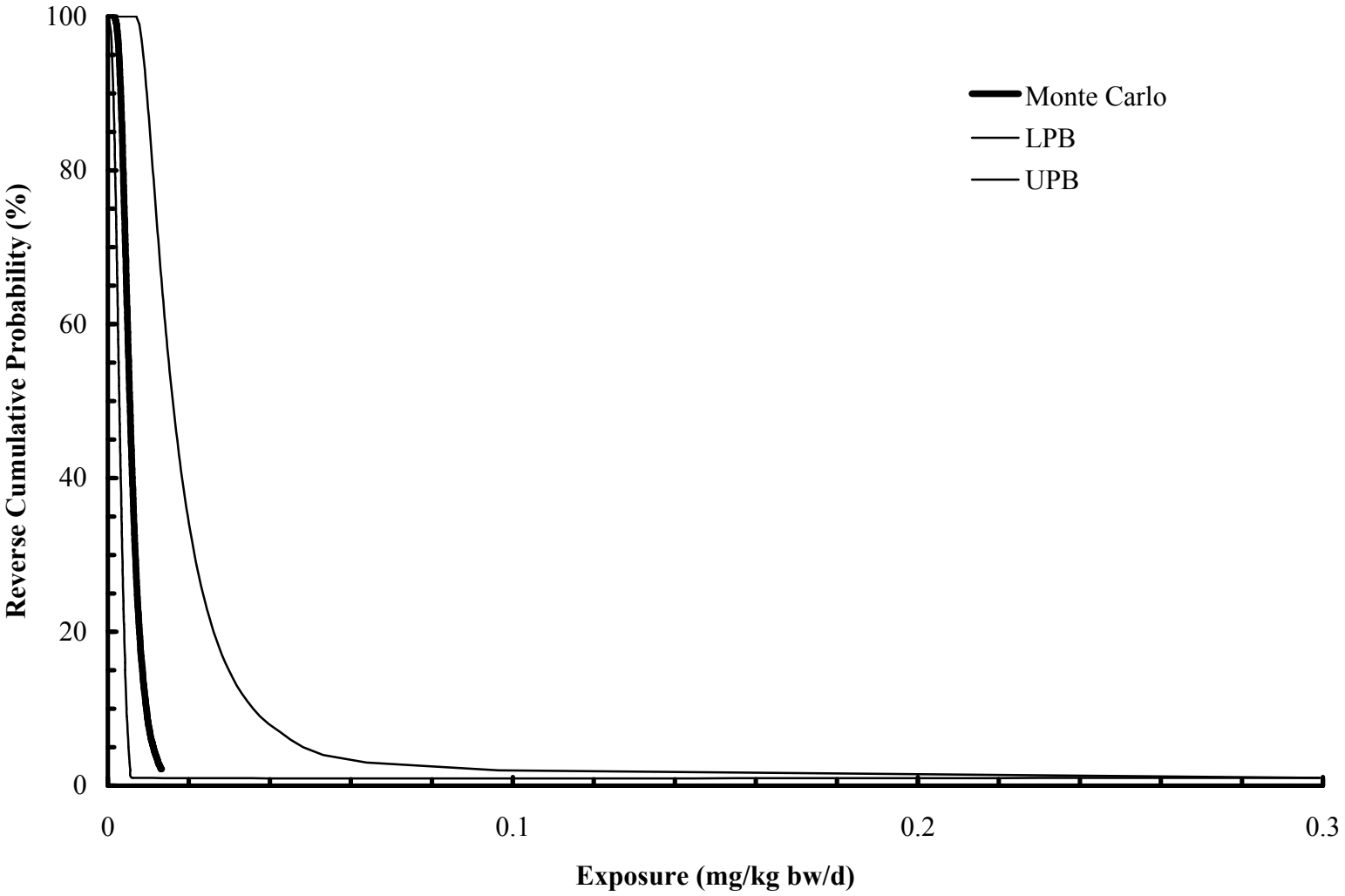

**Figure H3-25. Reverse cumulative probability distribution of total daily intake rates of total PCBs by small piscivorus birds in the reference areas, Calcasieu Estuary.**

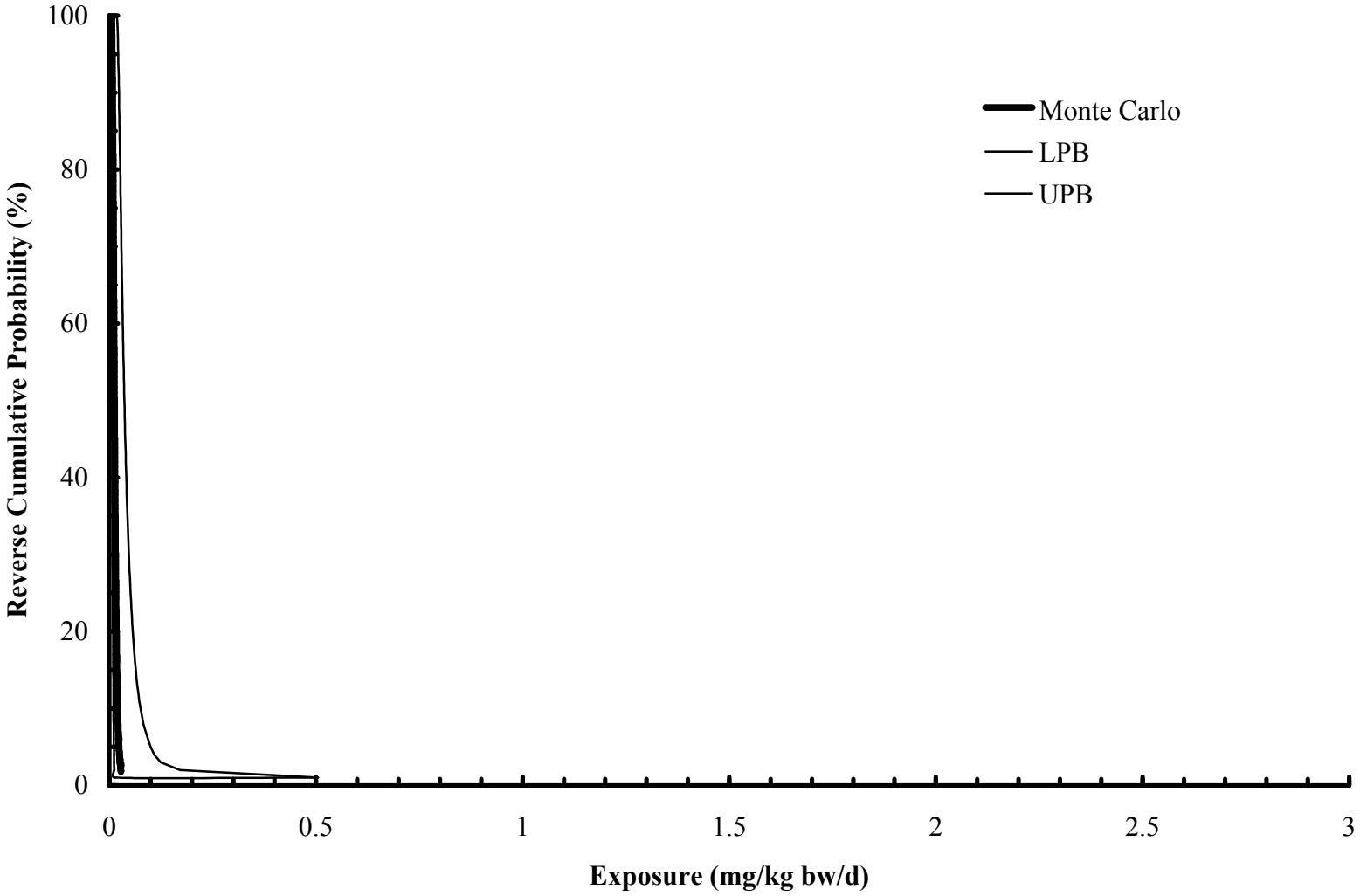

**Figure H3-26. Annual geometric mean concentration of Aroclor 1254 in fish fillet from Bayou d'Inde (bars represent minimum and maximum concentrations).**

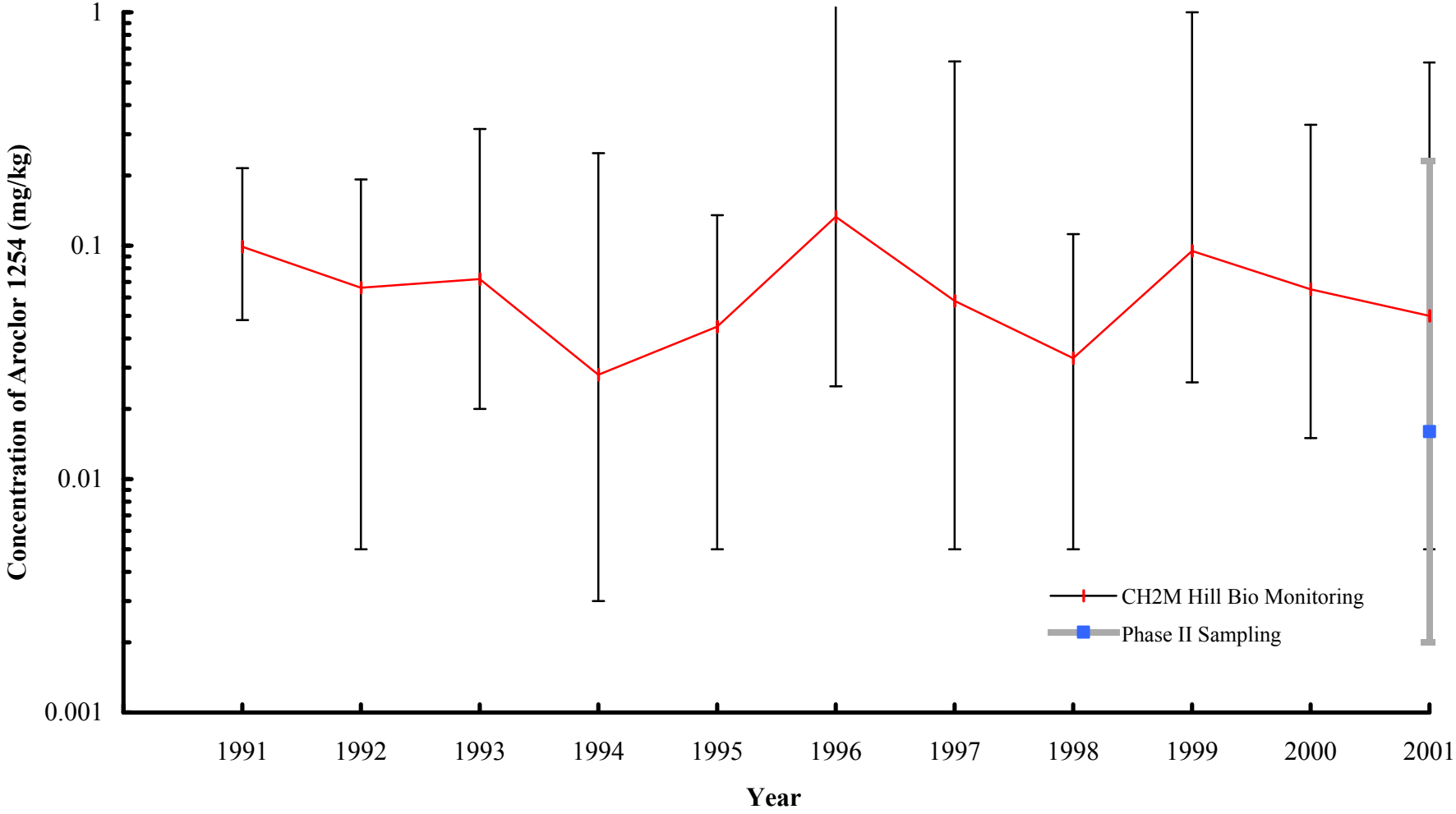

**Figure H3-27. Annual geometric mean concentration of Aroclor 1254 in fish fillet from the Upper Calcasieu River (bars represent minimum and maximum concentrations).**

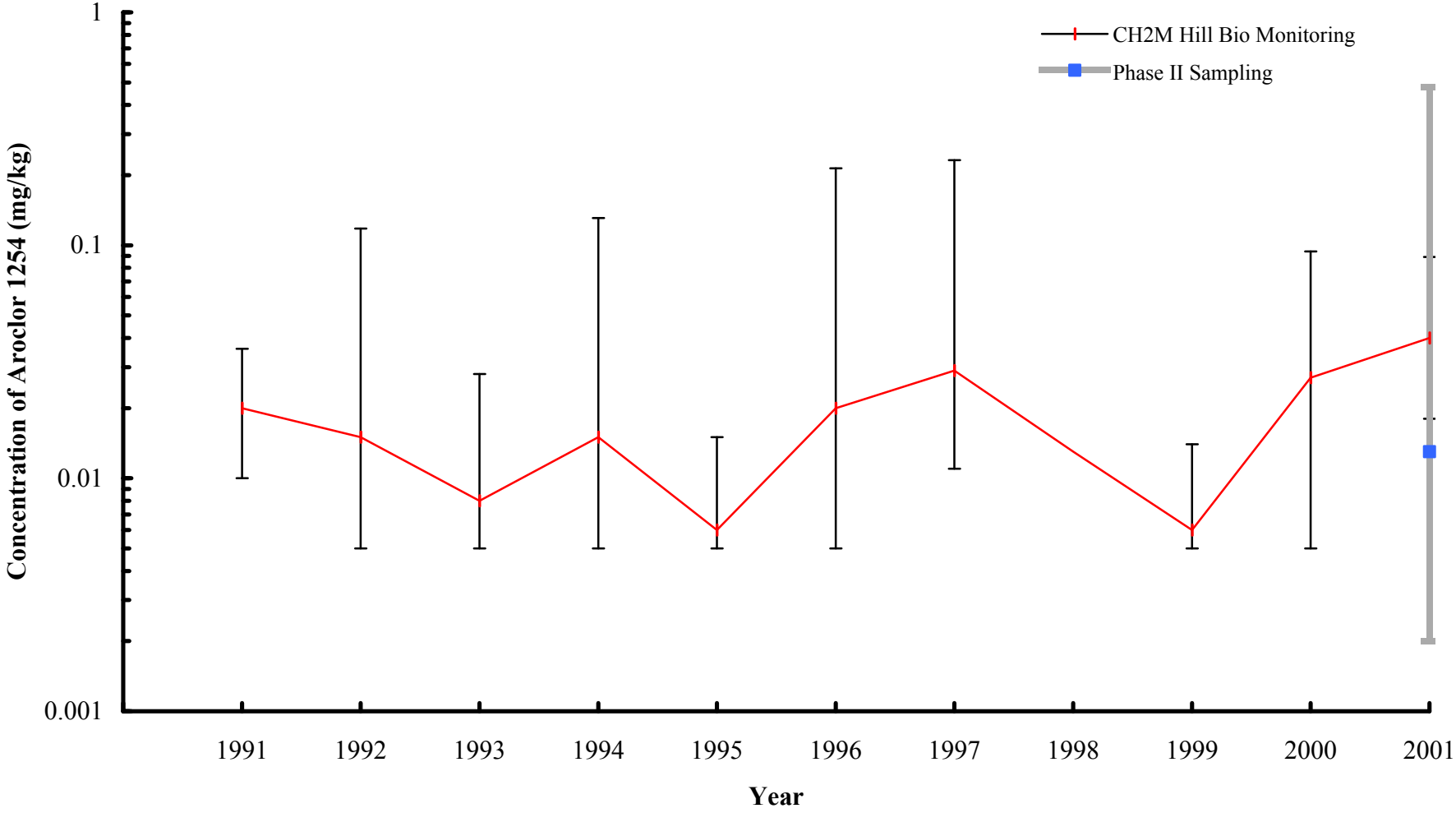

**Figure H3-28. Annual geometric mean concentration of Aroclor 1254 in fish fillet from the Middle Calcasieu River (bars represent minimum and maximum concentrations).**

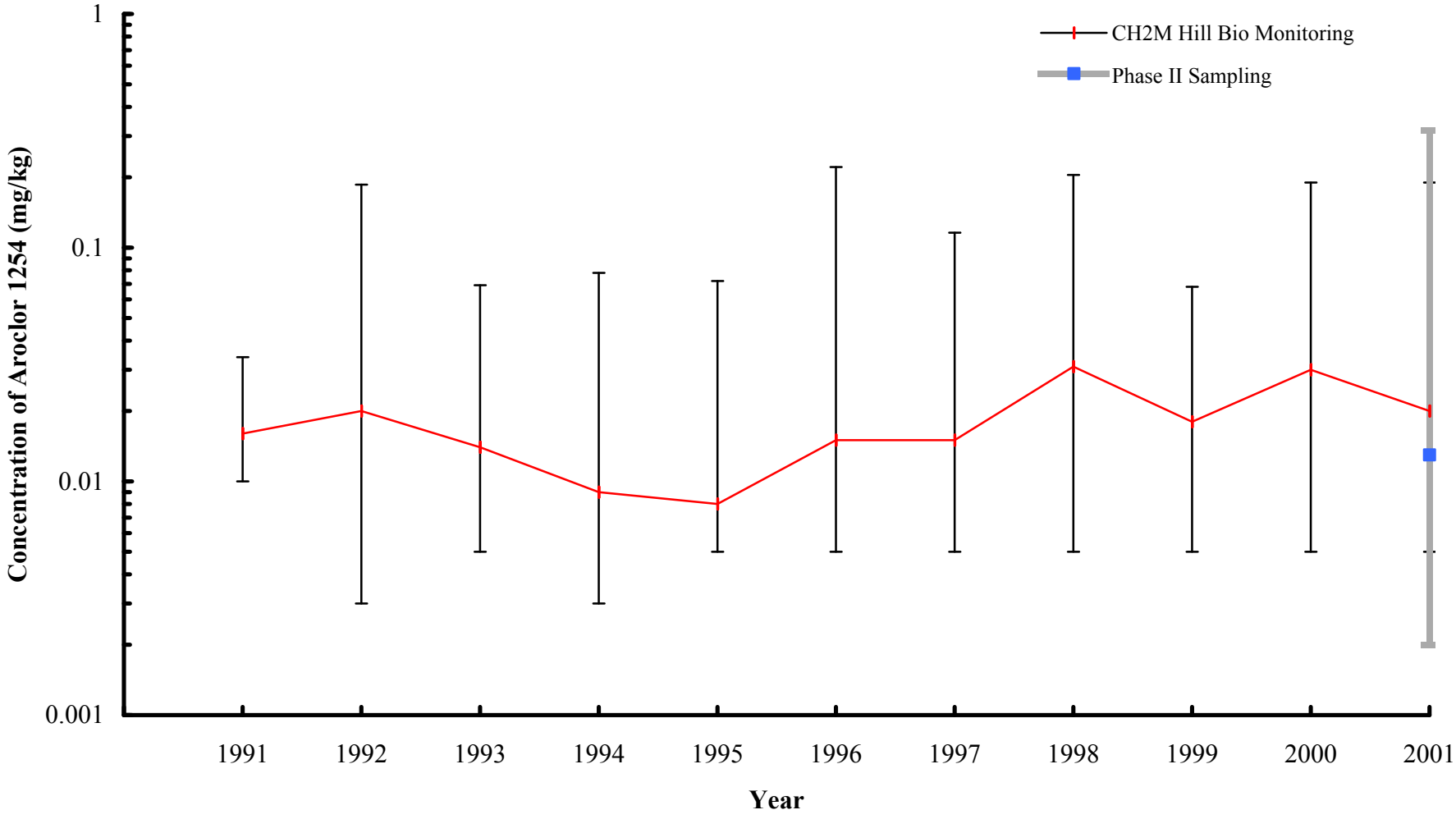

**Figure H3-29. Annual geometric mean concentration of Aroclor 1254 in fish fillet from the reference areas of the Calcasieu Estuary (bars represent minimum and maximum concentrations).**